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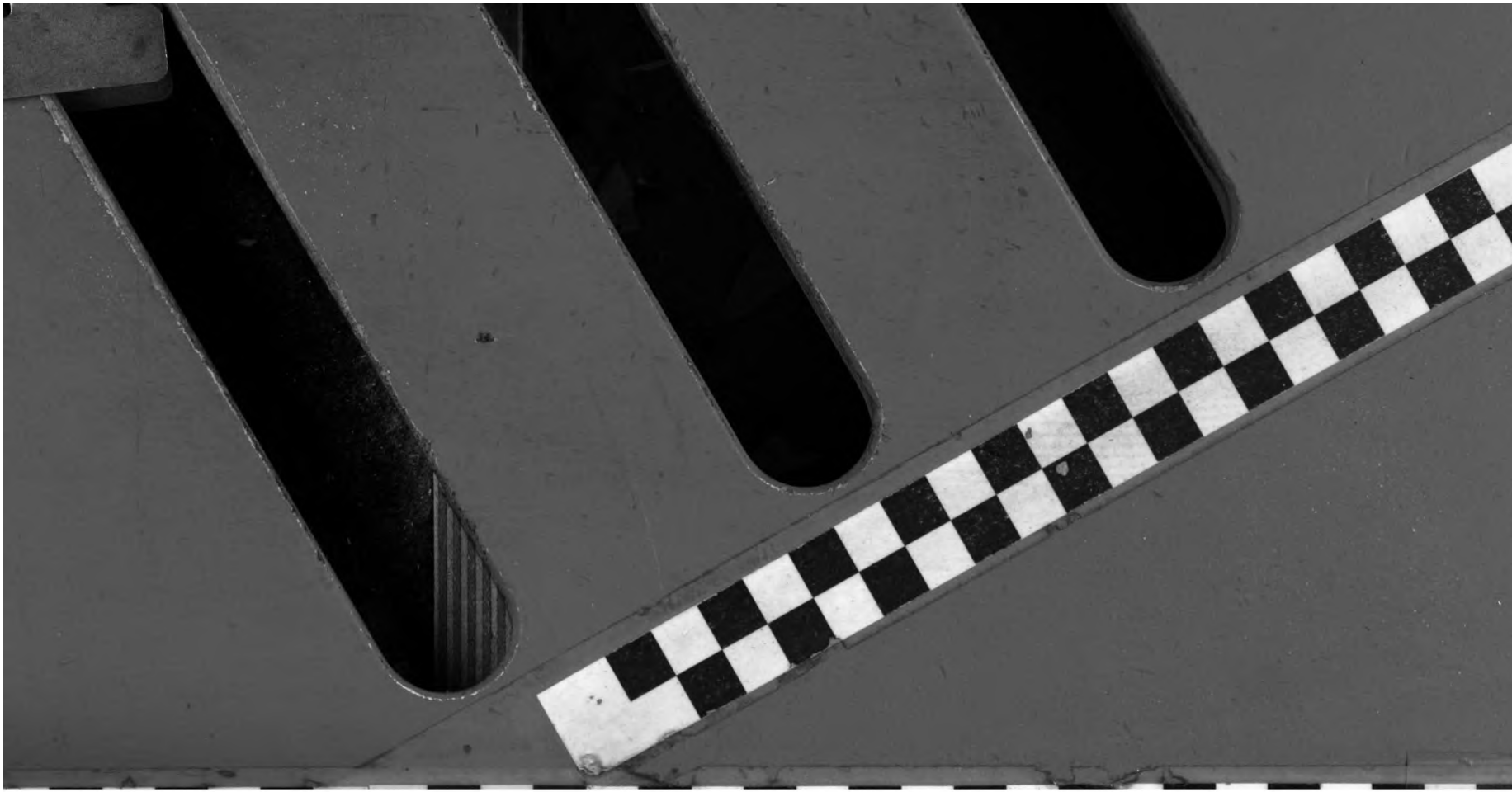

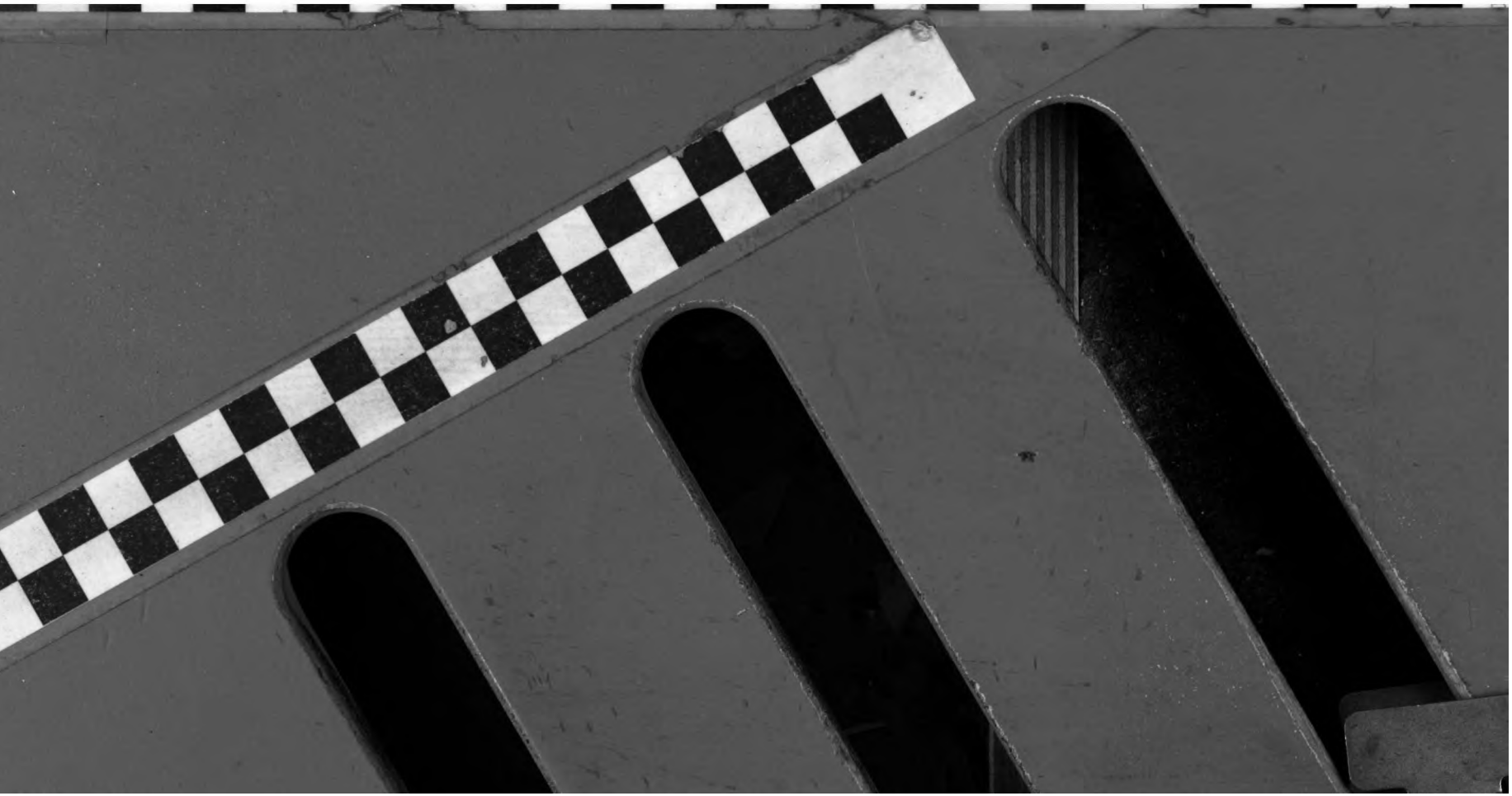

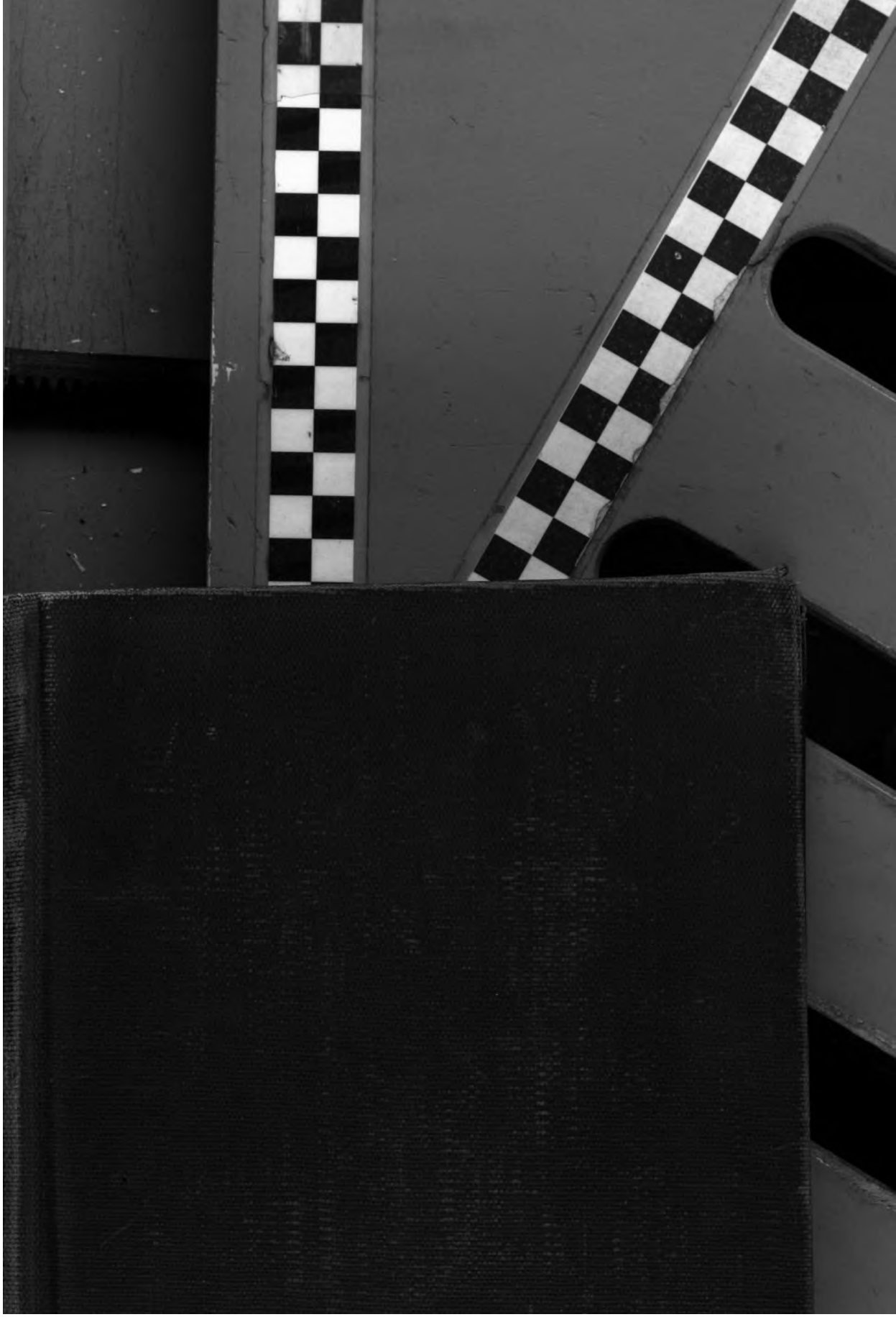

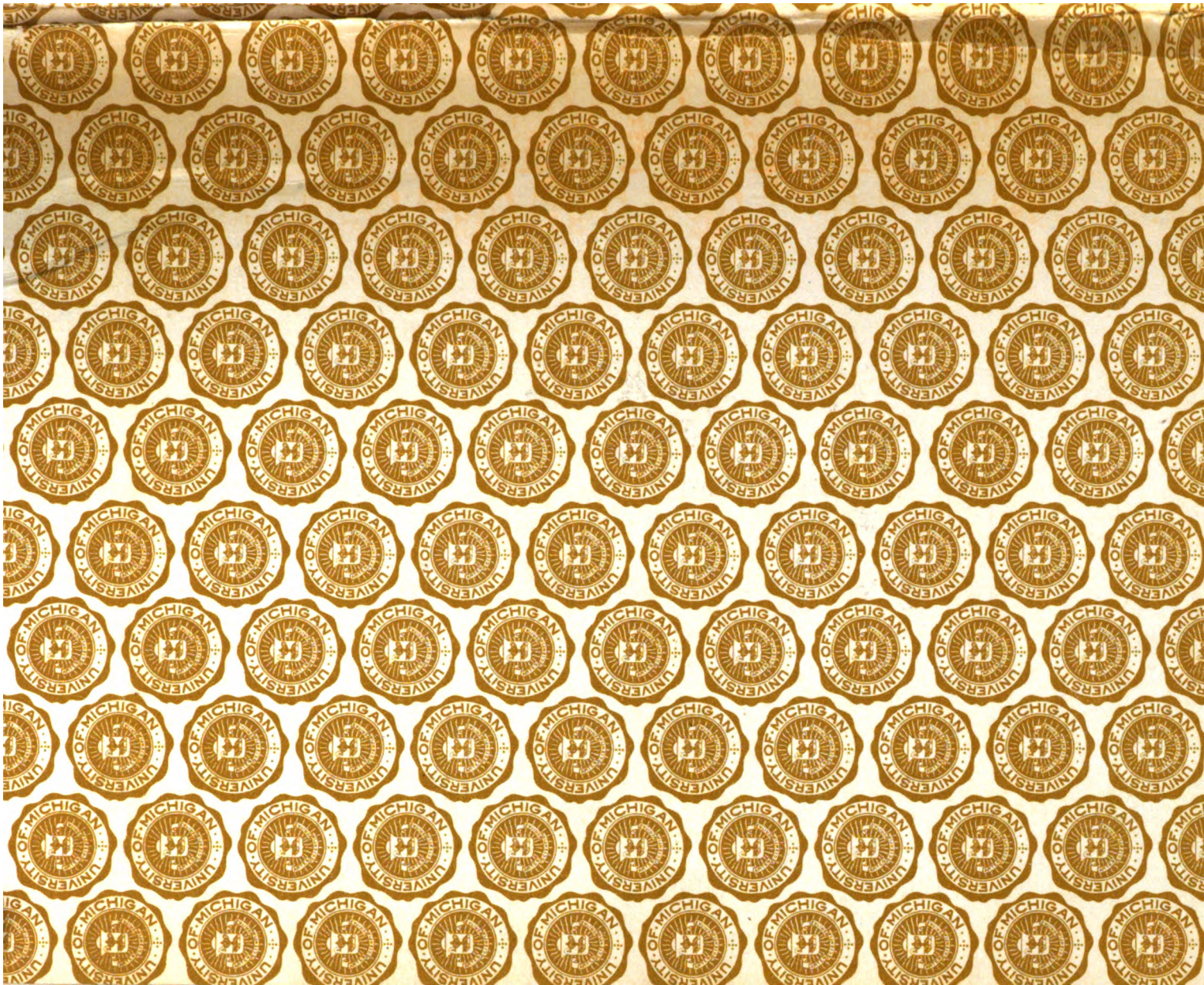
UNIVERSITY OF MICHIGAN

TEXT-BOOK OF NERVOUS DISEASES

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OF

NERVOUS DISEASES

FOR

PHYSICIANS AND STUDENTS

BY

PROFESSOR H. OPPENHEIM

OF BERLIN

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DISEASES OF THE BRAIN

ANATOMY AND PHYSIOLOGY OF THE BRAIN. LOCALISATION

Discussion of diseases of the brain should be prefaced by a short account of its anatomy and physiology, to bring together those facts with which the physician must be familiar before he can enter upon the study of brain pathology.

The Membranes of the Brain

The *dura mater* forms the inner periosteum of the skull. It may be fairly easily stripped in most places from the inner table of the skull of the adult. At the base of the skull it is attached more firmly to the bones.

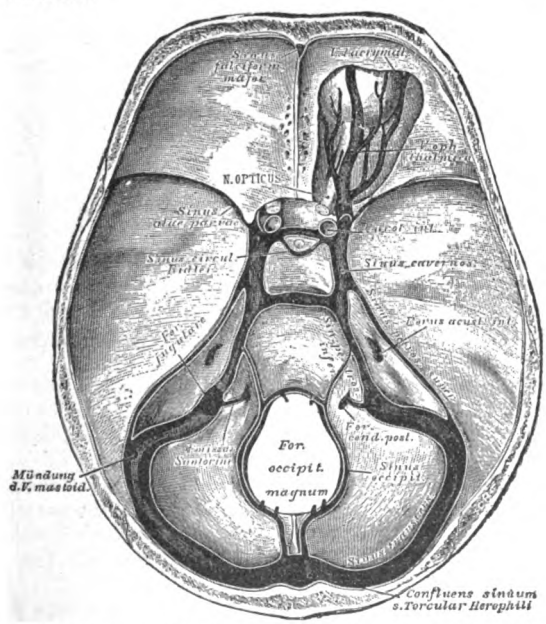


FIG. 251.—The sinuses of the dura mater. Bird's-eye view. (After Heitzmann.)

The dura divides into two layers where it forms the *sinuses*, and also where the nerves pass through it. Within the cranial cavity it gives rise to projecting processes. The great vertical

process, the *processus falciformis major* (falx cerebri), stretches from the crista galli to the internal occipital protuberance, and is attached with its two layers to the lateral margins of the sagittal sulcus. It passes inwards between the hemispheres, so that its lower margin is only 2 mm. distant from the upper surface of the corpus callosum. Above, the *superior longitudinal sinus* is surrounded by the two layers and by the dura lining the sagittal sulcus, while the under margin encloses the *inferior longitudinal sinus*. On each side of the middle line, close to the superior longitudinal sinus, are the hollow spaces (parasinoidal spaces) into which the cerebral veins open before they reach the sinus. The *processus falciformis minor* (falx cerebelli) stretches from the internal occipital protuberance to the foramen magnum, and is placed between the two cerebellar hemispheres. The outer margin, which is attached to the occipital crest, here forms the *sinus occipitalis*.

The transverse process, the *tentorium cerebelli*, lies between the inferior surface of the occipital lobes and the upper surface of the cerebellum. It is inserted into the transverse lines of the occipital bone and forms here part of the *transverse sinus*; also to the upper edge of the petrous

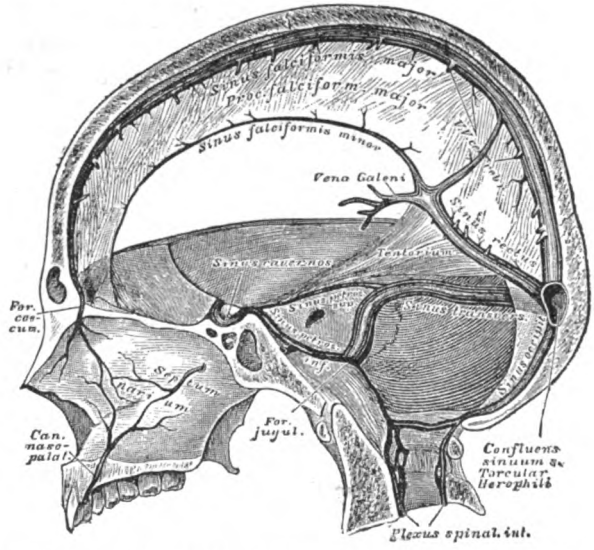


FIG. 252.—The sinuses of the dura mater. Lateral view. (After Heitzmann.)

bone, where it includes the *superior petrosal sinus*. The anterior free border is deeply concave (incisura tentorii). The median part of the tentorium lies upon the monticulus cerebelli.

Figs. 251 and 252 demonstrate the position and course of the sinuses of the dura mater, so that further description of these is not necessary. As regards the junction of the sinuses with the intra- and extra-cranial veins, see Fig. 253.

Processes of dura mater, the dural sheaths, accompany the emerging cranial nerves. The optic nerve is accompanied by its dural sheath as far as the eyeball, while that of the auditory and facial nerves penetrates with these nerves into the internal auditory meatus, or even reaches into the facial canal.

The branches of the middle meningeal artery run on the outer surface of the dura, and in the well-known furrows on the inner surface of the skull. The veins of the dura anastomose through numerous branches with those of the skull. The nerves of the dura are branches of the sympathetic fibres which accompany the arteries, and there are also a few twigs from the fifth nerve (n. spinosus and n. tentor cerebelli).

The histological structure of the dura mater has been most fully described a few years ago by Nose (*Obersteiner*, viii.).

The narrow space between the dura mater and the arachnoid—the subdural space—contains only a little fluid. The *cerebro-spinal* fluid is found in the sub-arachnoid space and in the ventricles.

The arachnoid mater is united in many places with the pia by the fine trabeculæ and membranes which form the subarachnoid tissue. Thus one cannot speak of one subarachnoid space. On the

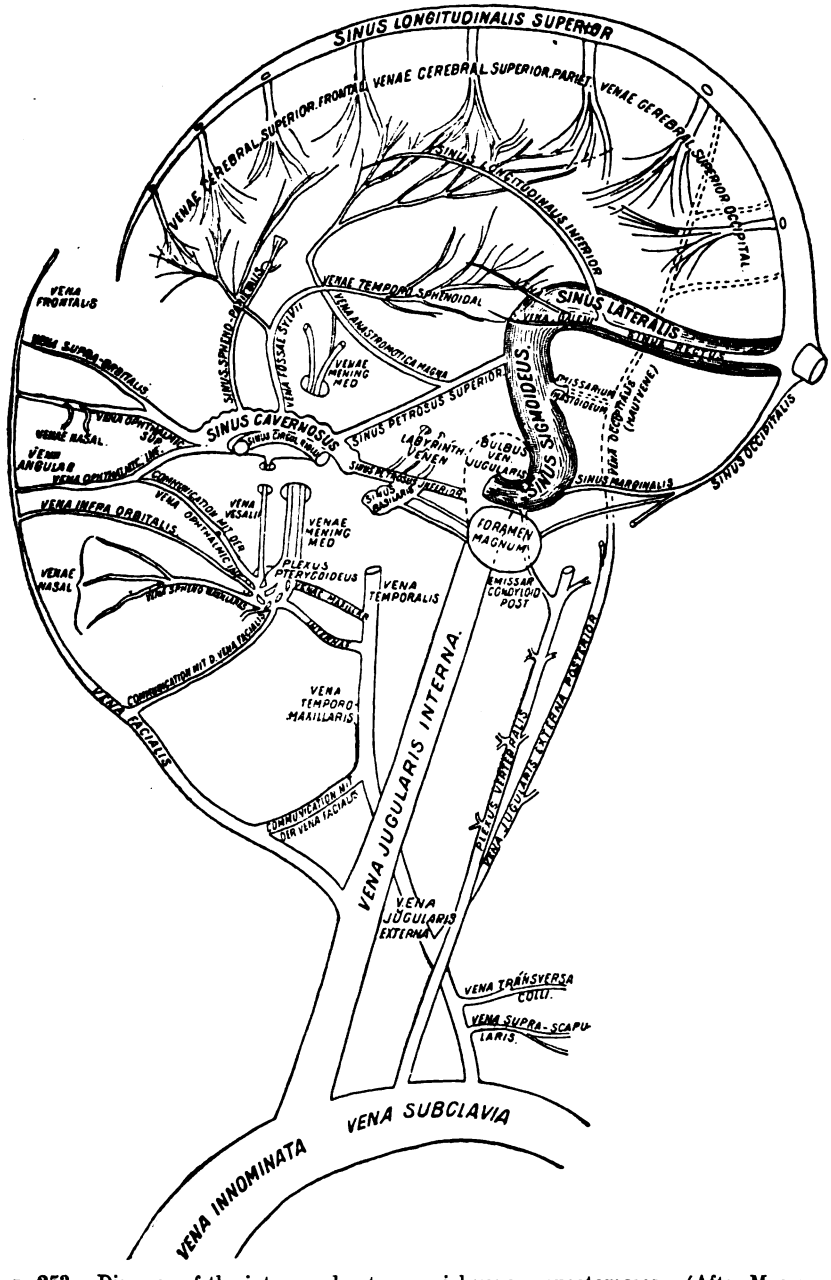


FIG. 253.—Diagram of the intra- and extra-cranial venous anastomoses. (After Macewen.)

contrary, there are a number of smaller and larger inter-communicating subarachnoid spaces. At the convexity the arachnoid and the pia form a single membrane. The arachnoid stretches over the fissures, while the pia covers all the outer surface of the brain and lines the bottom and sides

of the fissures. At the base of the brain the arachnoid is separated from the pia for a large extent, so that large subarachnoid spaces (*cisternæ subarachnoidales* or *subarachnoidal sinuses*) are formed. In a similar manner the *cisterna magna cerebello-medullaris* is formed by the arachnoid bulging over the space between the dorsal surface of the medulla oblongata and the hinder part of the lower surface of the cerebellum and up to the superior vermis, and it does not penetrate into the space between the inferior vermis and the tela choroidea. The cord is surrounded throughout its whole extent by a wide subarachnoid space. At the base of the brain there are formed a cisterna chiasmatis, intercruralis, fossæ Sylvii, etc.

The larger blood-vessels of the brain are situated within the subarachnoid spaces.

The *subarachnoid tufts or Pacchionian bodies* are club-shaped, cauliflower-like outgrowths of the arachnoid, which penetrate the substance of the dura mostly in the region of one of its sinuses or of the parasinoidal spaces, and may so thin it that they form the well-known impressions on the inner surface of the skull. The tufts can even grow through the bone. The venous blood spaces are separated by a thin layer of dura mater. The tissue of these tufts is a continuation of the subarachnoid tissue. It has been proved experimentally that an overflow of serous fluid from the subarachnoid spaces may take place from these tufts into the sinuses of the dura, if the pressure in these is low. The arachnoid also forms sheaths to the nerve roots.

The subarachnoid spaces communicate with the ventricles. The openings of communication—the *foramen of Magendie*, etc.—are at the fourth ventricle, at its posterior end. These are one fairly large oval aperture in front of the calamus scriptorius, and two other lateral ones at the apices of the lateral recess. A recently expressed objection to this opinion may be disregarded here.

The *pia mater* is closely adapted to the surface of the brain and penetrates into the ventricles as the *tela chorioidea*. The tela chorioidea superior (velum interpositum) enters the cerebrum through the transverse fissure, and bridges across the third ventricle.

The choroid plexus which accompanies it is continued through the foramen of Monro into the lateral ventricle. The two veins of Galen run in the tela chorioidea and unite to form the unpaired or great vein of Galen at the posterior pole of the pineal gland. The tela chorioidea inferior lies between the ventral surface of the cerebellum and the dorsal surface of the medulla oblongata.

The pia receives sympathetic nerve fibres, which spring from the plexus surrounding the vessels of the circle of Willis, also branches from several cranial nerves.

Convolutions and Fissures of the Surface of the Brain

The position and course of the gyri and sulci is to be seen in Figs. 254, 255, and 256.[1]

They may be most easily recognised on the surface of the brain if one starts at the *fissure of Sylvius and the central sulcus*. The *fissure of Sylvius*, in which there are to be distinguished one long posterior and two short anterior limbs, passes from before backwards and from below upwards, and separates the frontal lobe, central convolution and part of the lower parietal lobe from the temporal lobe.

At the bottom of the fissure is situated the island of Reil, consisting of several small convolutions.

The part of the brain which is superficial to the island of Reil is known as the *operculum*. This includes the foot of the central convolution, the posterior part of the third frontal convolution, and the segment of the lower parietal lobe which lies between the interparietal fissure and the fissure of Sylvius.

In the operculum there begins an important fissure which is always well marked, and which runs from before backwards and from below upwards to the margin of the brain, viz., the *fissure of Rolando* or *central sulcus*. It is bounded in front and behind by the *anterior* and *posterior central* convolutions.

[1] See literature in Oppenheim: "Die Geschwülste des Gehirns," 2nd ed., in Nothnagel's "Handbuch," ix. 2; Kocher, in Nothnagel's "Handbuch," Bd. ix. 3; Waldeyer, *D. m. W.*, 1901.

The anterior central convolution forms the posterior limit of the frontal lobe, from which it is practically separated by an inconstant fissure, the *sulcus præcentralis*.

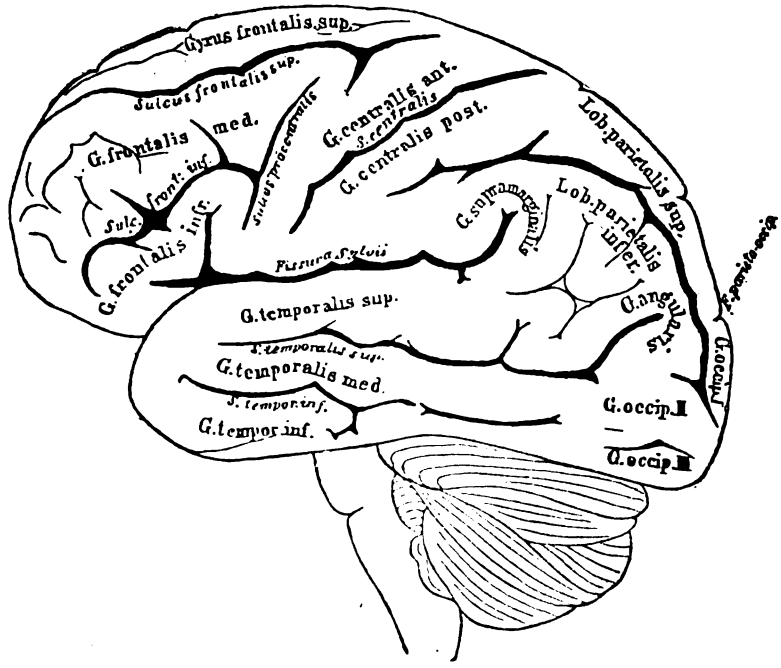


FIG. 254.—Lateral view of the brain. (After Ecker.) Gyri and sulci.

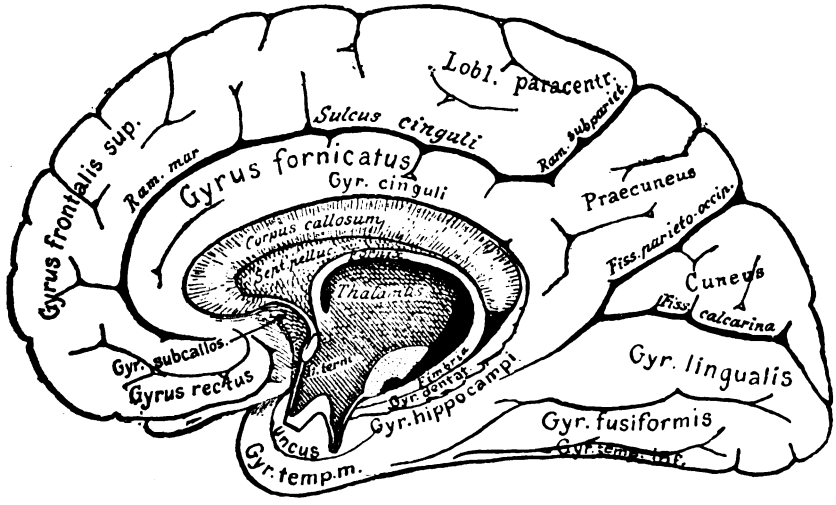


FIG. 255.—View of the median surface of the hemisphere. (After Edinger.)

The *frontal lobe* is divided by two fissures into three convolutions running in a sagittal direction. They are mostly connected to one another by means of annectant convolutions. The lowest is known as the

third. The most posterior part of the third frontal convolution, lying behind the anterior ascending limb of the fissure of Sylvius, is known as the pars opercularis. Next to it comes the pars triangularis and then the pars orbitalis of the frontal lobe.

The *temporal lobe* is divided into upper, middle, and lower temporal convolutions by fissures which run parallel with the fissure of Sylvius.

The *parietal lobe* passes from behind into the posterior central convolution. It is divided into an upper and a lower parietal lobe by a curved, discontinuous fissure, the *interparietal sulcus*. The upper lobe

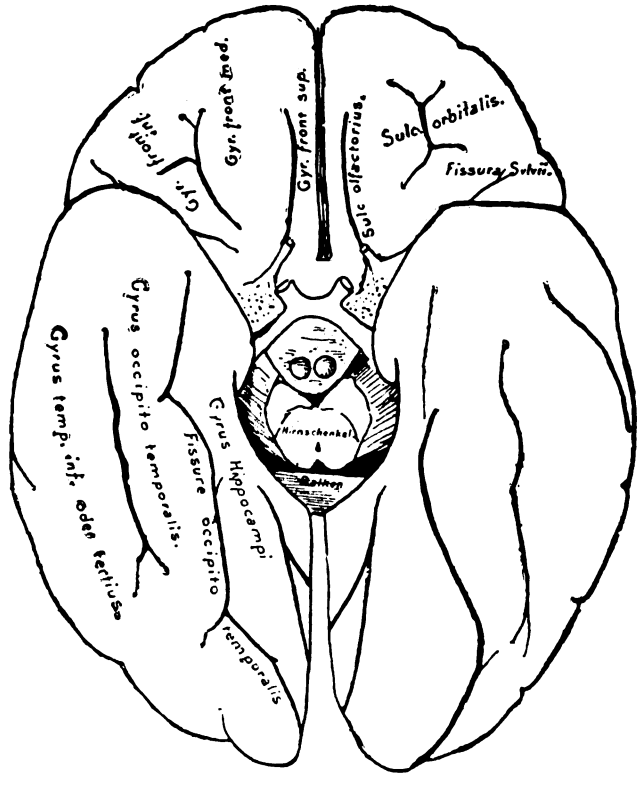


FIG. 256.—Convolutions at the base of the brain. (Edinger, drawn after Ecker.)

is directly continuous with the posterior central convolution; the lower consists of a *gyrus supramarginalis*, which bends round the posterior end of the fissure of Sylvius, and the *gyrus angularis*, bending round the upper temporal fissure. The latter is continued posteriorly into the gyrus parietalis posterior. The interparietal fissure may be divided into three parts, which are sometimes distant from one another. The most anterior is also known as the fissura retrocentralis inferior, the most posterior as the sulcus occipitalis anterior or perpendicularis.

The *occipital lobe* is divided from the parietal lobe by the anterior *occipital fissure*, as also on the median side by the *parieto-occipital fissure*.

On the mesial wall of the hemisphere (Fig. 255), immediately above the corpus callosum, is to be found the *gyrus fornicatus*, the upper boundary

of which is formed by the *sulcus callosomarginalis* or *s. cinguli*. It is continued posteriorly into the *præcuneus*, which corresponds with the mesial surface of the parietal lobe. In front of it lies a part of the brain which forms the direct continuation of the central convolution, viz., the *paracentral lobe*. The præcuneus is bounded posteriorly by the parieto-occipital fissure.

This fissure and the *calcarine fissure* which forms a sharp angle with it, cut off a wedge-shaped area on the mesial wall of the hemisphere, the *cuneus*. The *gyrus fornicatus* surrounds the splenium of the *corpus callosum* in an arc, and forms the *gyrus hippocampi*, which is the uppermost convolution of the mesial surface of the temporal lobe and terminates in its most anterior part as the *gyrus uncinatus*. A convolution situated below the calcarine fissure, the *lobus lingualis*, is directly continuous forwards with the gyrus hippocampus. The *gyrus fusiformis* lies next to the lobus lingualis, and is separated by the inferior temporal sulcus from the third temporal convolution. The convolutions at the base of the brain are shown simply in Fig. 256.

Relation of the Skull to the Surface of the Brain

This is shown in Figs. 257 and 258.

The frontal bone covers the greater part of the frontal lobe and the whole of the lowest convolution. The bases of the frontal convolutions lie beneath the parietal bone.

The central convolutions, the parietal lobes, and a part of the occipital lobe are situated under the parietal bone. The parietal eminence practically corresponds to the lower parietal lobe, or gyrus supramarginalis. The temporal bone covers the greater part of the temporal lobe, the squamosal suture lying with the highest point of its convexity over the fissure of Sylvius. This fissure extends, according to Waldeyer, in an oblique, slightly ascending direction, from the pterion or Sylvian point to the parietal eminence (Fig. 258).

The Sylvian point lies under the spheno-parietal suture, near its posterior end. The anterior horizontal ramus runs forwards, corresponding fairly closely to the sphenoparietal suture. The anterior ascending ramus at the Sylvian point forms a right angle with the posterior horizontal ramus, and crosses the lower end of the coronal suture at a sharp angle (Waldeyer). The point of division of the fissure of Sylvius into anterior and posterior limbs is to be found 4-4·5 cm. above the mid-point of the zygomatic arch (Merkel).

The fissure of Rolando has an almost constant relation to the coronal suture, its lower end lying about 28, its upper about 48-55 mm. behind it or behind the bregma (Fig. 258). Since, however, this suture is not always to be felt, it is necessary to employ other methods which allow the various convolutions and fissures to be determined without reference to the cranial sutures.

In order to find the upper end of the central or Rolandic fissure, a line is taken, according to Thane, Horsley, and Dana, in the middle of the skull or the sagittal suture between the root of the nose (nasion) and external occipital protuberance (inion). This is bisected and the upper extremity of the fissure of Rolando is found about 2 cm. behind this point. It is more exact to take the line connecting the glabella with the inion,

some 55·7 per cent. from the glabella. The fissure runs from this point forwards and downwards and forms an angle of 67° (or between 60° and 70°) with the sagittal line. Wilson has invented an instrument for these measurements.

In order to determine the lower extremity of the fissure of Rolando, Poirier first marks the upper border of the zygomatic process of the temporal bone and from this base line draws a perpendicular line which ascends exactly in front of the tragus, between it and the posterior end of the temporo-maxillary joint. The looked-for point lies 7 cm. above the auditory meatus. Lucas Championnière uses another method

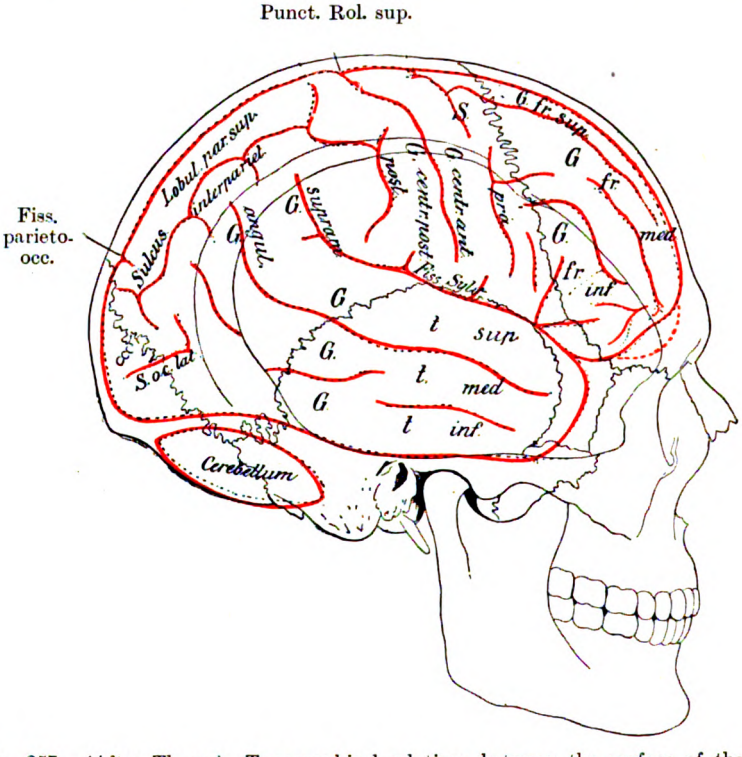


FIG. 257.—(After Thane.) Topographical relations between the surface of the brain and the skull.

for the determination of this point. He draws a horizontal line backwards for 7 cm. from the orbito-temporal angle, and at the end of this line he draws another perpendicular one. The point will be found 3 cm. along this line. On the child's skull the lower end point is to be found by connecting the external auditory meatus to the sagittal suture by a vertical line, and measuring off 15 mm. below its mid-point.

Bennett and Godlee, following Reid and Bergmann, employed another method for the projection of the central fissure on to the surface of the skull. We have tested this method and found it of great use. A perpendicular line is drawn from the sagittal line (nasion to inion) at the anterior margin of the external auditory meatus, and about two inches behind it a second parallel line is drawn, which passes across the posterior

margin of the mastoid process. The point at which this latter line meets the sagittal line is the upper end of the central sulcus. The lower end is found on the anterior vertical line about two inches above the external auditory meatus or two inches above its upper margin. Various instruments (cyrtometer, encephalometer, cranioencephalometer) have been constructed for these purposes, *e.g.* one made by Détert in Berlin on Köhler's plan. Others have been devised by Krönlein, Horsley, and Kocher. Kocher's instrument consists of a horizontal bar, as long as the base line which unites the glabella with the external occipital protuberance, and is fitted to the skull. This is connected with a pliable

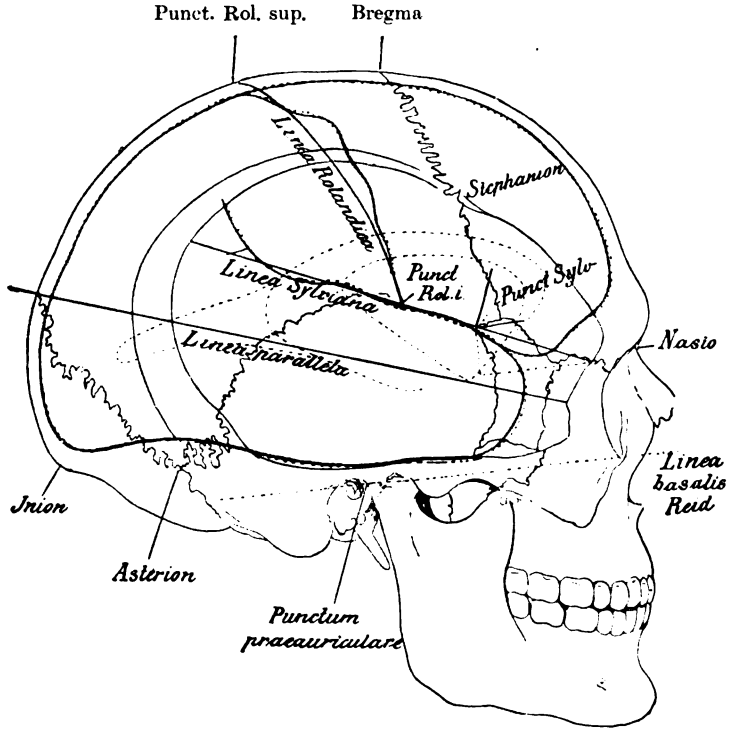


FIG. 258.—(After Thane.) Topographical relations between the surface of the brain and the skull.

sagittal steel band. A third band, also of pliant steel, may be moved on the sagittal band, and may be turned in all directions on a plate with circular divisions. These instruments may practically always be dispensed with and may be replaced by exact measurements by means of narrow strips of adhesive plaster which are adapted to the skull in the manner described above.

Kocher regards the recognition of the precentral fissure as the most important point in cranio-cerebral topography. He gives the following directions: find the middle of the sagittal line between the glabella and the external occipital protuberance, and place upon this point the bevelled scale of Kocher's cyrtometer, so that it forms an angle of 60° open in front, with this sagittal line. The bevelled scale then lies over the precentral fissure for its whole length, except the uppermost part, which covers the anterior central convolution.

When the position of the central fissure has been ascertained, it is not difficult to project the different parts of the motor area on to the skull. The anterior central convolution begins below the anterior lower angle of the parietal bone.

The three outer occipital convolutions correspond to the upper part of the occipital bone, and a great part of the cerebellum corresponds to the lower. An anterior portion of it lies beside the mastoid portion of the temporal bone (Fig. 257). The third temporal convolution and also the anterior part of the gyrus fusiformis and lingualis, lie on the anterior surface of the petrous bone, and the cerebellum lies on its posterior surface. The rest will be seen from Figs. 257 and 258.

Chipault has recently invented a simple method for projecting the cerebral fissure on to the outer surface of the skull. Berry and Shepherd also describe a special method (*Brit. Med. Journ.*, 1904).

The temporal muscle covers the whole of the temporal lobe, or rather its convexity, and the Sylvian fissure and the whole of the lower frontal convolutions are also concealed below it (Merkel).

With regard to all these statements, however, it should be borne in mind that the topographical relations exhibit individual differences which depend chiefly on the form of the skull. Thus Foriep distinguishes two types of the position of the brain within the skull. viz., the *frontipetal* and the *occipitopetal*. The former is found in short and high skulls, where all the fissures and convolutions lie further forwards, whilst in the occipitopetal type, in long, low skulls, they are displaced backwards and downwards.

Schwalbe has pointed out the interesting fact that the projection of the brain, especially of the parts covered by muscles, corresponds with the elevation on the outer surface of the skull. This in itself affords a certain guide to the position of the individual convolutions. Thus he finds the protuberance of the cerebellum at the occipital bone, a triangular part or protuberance of the inferior frontal gyrus at the frontal bone, or the frontal and parietal bone, and the temporal convolutions produce an eminence or protuberance of the temporal bone, etc.

Histological Structure of the Cerebral Cortex

In many parts of the cerebral cortex it is possible with the naked eye to recognise on the surface of the section, laminæ parallel to the surface. This is most distinct and constant in the case of the calcarine fissure, where a whitish line separates an outer grey from an inner yellow-grey layer. This line of Vicq d'Azyr corresponds in other places to the less distinct line of Gennari or (the outer) line of Baillarger.

Microscopical examination of the cerebral cortex does not show a similar structure throughout, but it presents a general type, consisting of an arrangement of nerve-cells (and fibres) in which we can distinguish a series of layers, not, it is true, sharply separated from each other, but characterised by some predominant type of cell (Figs. 261, 262, and 263).

Below the pia mater there is a layer of neuroglia, which contains only a few *small cells*. At their outermost limit a number of nerve fibres run parallel with the surface—the *tangential fibres*. In the deeper parts of this layer, numerous *round cells* are seen. The *small pyramidal cells* compose a second layer. They are thus named from their pyramidal shape, the apex of the pyramid being directed outwards, and its base sending out an axis-cylinder process (Fig. 260). This layer gradually passes into that of the *large pyramidal cells*. This layer is characterised by successively arranged large pyramidal cells, which increase in size from without inwards. In addition to these there are thick layers of nerve fibres, which cut through at right angles.

The deepest layers contain, in the region of the central convolution, and especially of the *paracentral lobule* and the *anterior central convolution*, very large examples of ganglion cells, viz., the *giant cells of Betz* (see Fig. 264). A layer of small cells follows on the large pyramidal cells, and numerous *spindle cells* are found at their lowest boundary. An attempt has been made to mark off the limits of this layer as a special spindle-cell layer or one of polymorph cells. In addition a number of small polygonal cells are scattered through all the layers of the cortex. Thus we may distinguish four, five, or, according to Brodmann, six layers in the cerebral cortex.

Meynert found in some regions a five-, and in others an eight-layered type. Schlapp (*A. f. P.*, xxx.) speaks of a five-layered and a seven-layered type, and is inclined to bring the arrangement into relation to the function of the cortex, an attempt which has already been made by several writers, and which has been continued during the last few years with special success by Vogt-Brodmann [1] and Campbell.[2]

Brodmann proposes the following terms for the layers (compare Figs. 262-263): 1. lamina zonalis (molecular layer or "cell-free cortical layer"); 2. lamina granularis externa (outer granular layer or small crowded pyramids); 3. lamina pyramidalis (layer of middle or large pyramids); 4. lamina granularis interna (inner granular layer); 5. lamina ganglionaris (deep large pyramids, Hammarberg's ganglion layer); 6. lamina multiformis (polymorph or spindle-celled layer).

This fundamental type, as Bolton has already shown and as Brodmann has confirmed, undergoes a transformation in an area of the occipital lobe delimited by the latter into the so-called *calcarine type of Brodmann*, where the lamina granularis interna is divided into three layers by the appearance of the lamina intermedia (Vicq d'Azyr or Gennari).

The dendritic processes of the ganglion cells, as well as the axis-cylinder processes and their accessory branches, form an essential part of all the layers. Besides these, fibres arising from the medullary layer run through the whole extent of the cortex.

Edinger differentiates the fibre bundles as follows: 1. radii, radiations of medullary fibres; 2. inter-radial network, consisting mostly of superficial parallel fibres; 3. super-radial fibrous network, and 4. tangential fibres. At the margin between the super-radial and the inter-radial network, the latter becomes condensed into Gennari's or Baillarger's lines, and in the calcarine fissure into Vicq d'Azyr's line. It is supposed that the former are practically formed from the lateral twigs of the nerve processes originating from the pyramidal cells. Bechterew has been able to mark off a strip composed of

FIG. 259.—Section through the grey cortex of the cerebrum near the calcarine fissure. *f.ca* = calcarine fissure. The white line is Vicq d'Azyr's line. (After Schwalbe.)

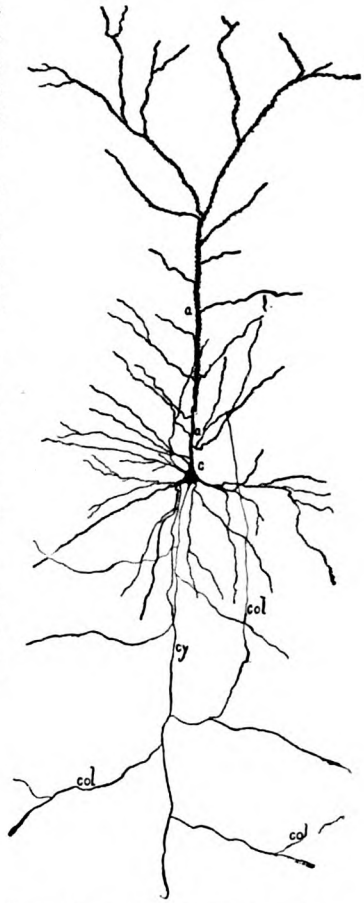


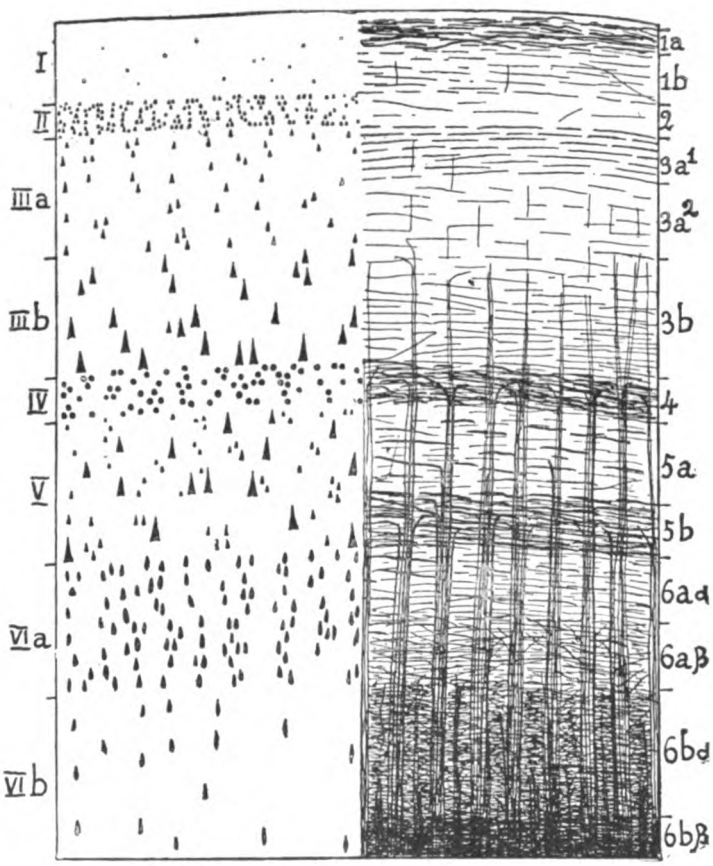
FIG. 260.—(After Dejerine.) Pyramidal cells of the human cerebral cortex, by Golgi's method. *cy* = axis-cylinder process; *c* = cell-bodies; *col* = collaterals of the axis-cylinder. Of the protoplasmic processes only the ascending one is specially marked (*a*).

medullary fibres in the inner marginal layer below the tangential fibres. There are also fibres in the deepest layer of the cortex which can be partly traced from one gyrus into a neighbouring one (association fibres). For the distribution and demarcation of the fibres according to Vogt's investigations see Fig. 263. Vogt has also shown that the distribution of the fibres

[1] *Journ. f. Psych.*, Bd. ii. *et seq.*; also *Anat. Anzeig.*, 1906, etc.
[2] "Histological Studies on the Localisation of Cerebral Function," Cambridge, 1905.

varies in the different areas of the cerebral cortex, and upon this fact he has based his topography. The investigations of Kaes[1] and others have shown that the number of medullated fibres in the cortex is materially increased after birth and even during the first half of life. During the first months of life only a few areas of the cortex show medullated fibres.

FIG. 261.—Diagram of the layers of the cerebral cortex. Region of the first occipital gyrus. (After Bevan Lewis and Gowers.)



FIGS. 262-263.—Diagrammatic representation of the arrangement of the fibres to the cells and laminæ of the cortex, in Brodmann's fundamental type.

I. = Lamina zonalis; II. = Lamina granularis externa; IIIa. = Pars medio-pyramidalis; IIIb. = P. magnopyramidalis laminæ pyramidalis; IV. = Lamina granularis interna; V. = Lamina ganglionaris; VIa. = Pars triangularis; VIb. = P. fusiformis laminæ multiformis; 1a = Pars superficialis; 1b = P. profunda laminæ tangentialis; 2 = Lamina afibrosa; $3a^1$ = Stria Kaesi-Bechterewi; $3a^2$ = Regio typica partis superficialis; 3b = Pars profunda laminæ suprastriatæ; 4 = Stria Baillargeri externa; 5a = Lamina intrastriata; 5b = Stria Baillargeri interna; 6aα = Lamina substriata; 6aβ = Lamina intermedia; 6bα = Lamina Meynerti; 6bβ = Substantia alba; 1*b*-3*b* = Supraradial; 5a-6b = Intraradial felt-work of Edinger.

[1] A work has lately been published which summarises his results: "Die Grosshirnrinde des Menschen," etc., Jena, 1907.

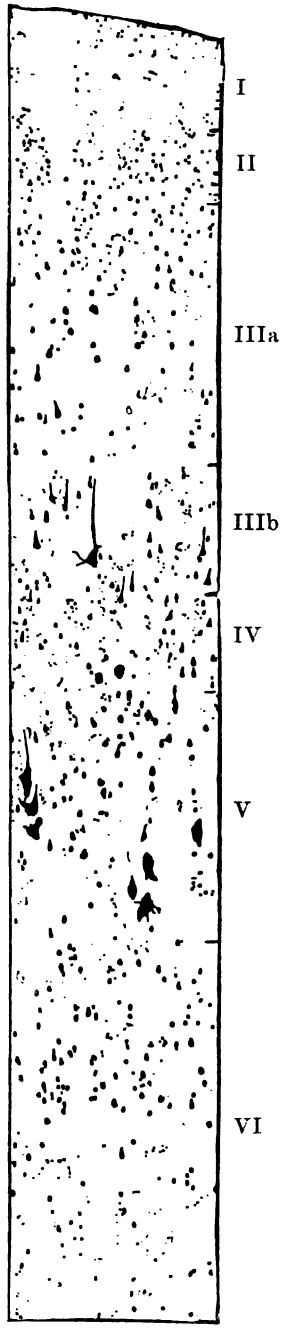


FIG. 264.—Section from the top of the anterior central convolution.

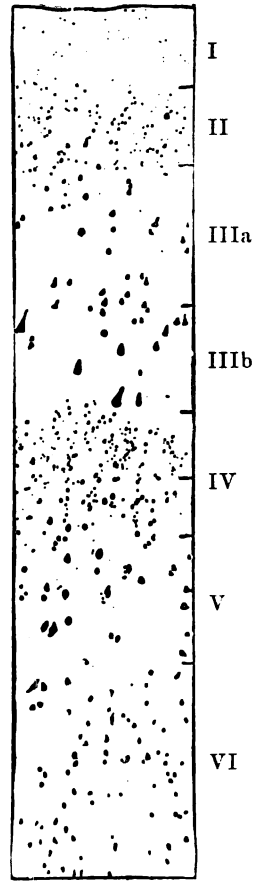


FIG. 265.—Section from the top of the posterior central convolution.

(After Vogt.)

I. = **Lamina zonalis**, marginal layer with few cells; II. = Lamina granularis externa; layer of small closely compacted pyramidal cells; III. = Lamina pyramidalis, layer of medium (IIIa.) and large (IIIb.) pyramidal cells; IV. = Lamina granularis int., Meynert's granular layer; V. = Lamina ganglionaris, layer of deep-lying pyramidal cells; VI. = Lamina multiformis, layer of polymorph or spindle cells.

Flechsig,[1] Monakow,[2] O. and C. Vogt,[3] Hösel.[4] and others have investigated the question of the time at which the medullated fibres appear in the different areas, but we cannot here examine their results. Further attempts to characterise and delimit the various areas of the cerebral cortex according to the condition and arrangement of the cells and nerve fibres have been made by Betz, Ramon y Cajal,[5] Nissl,[6] Passow, Kolmer, Bolton, and lately by Brodmann. They have established certain characteristics of the motor zones and the occipital lobe. In addition to the peculiarities of the anterior central convolution already mentioned, Ramon y Cajal has pointed out the absence of a distinctly differentiated granular layer, and the great breadth of the layers of the middle-sized and large pyramidal cells, etc., while Passow has shown that the super-radial and inter-radial fibre network in the anterior central convolution are very strongly developed. Brodmann has confirmed and supplemented Ramon y Cajal's statements by careful investigations. He and Vogt (compare Figs. 264 and 265) think that the anterior central convolution can be sharply differentiated from the posterior by its cyto-histological structure; its margin passes through the central sulcus, and this division corresponds to that established by Sherrington for function (see next chapter). Campbell has come to the same conclusions independently of Vogt. Flechsig has lately stated that there are characteristic differences between the anterior and posterior central convolutions in regard to the distribution and course of the fibres which are medullated at an early stage.

The *visual area* is, according to Ramon y Cajal, specially characterised by the appearance, in the area corresponding to the granular layer, of stellate cells with long descending axis-cylinders, etc. He also found a certain form of cells in the temporal lobe or in the first temporal convolution which he terms the acoustic form. The cyto-histological character of the visual region has been specially studied since then by Brodmann and Campbell. We cannot here discuss further minutiæ, but the investigations of Vogt and Brodmann lead us to hope that knowledge of the "myelo- and cyto-architecture" of the cerebral cortex will create a wider basis for the study of physiology and localisation.

Localisation in the Cerebral Cortex

See bibliography in Monakow: "Gehirnpathologie," II. Aufl., Wien, 1905; Nothnagel's "Handbuch," Bd. xi., and in Monakow: "Über den gegenwärtigen Stand der Frage nach der Lokalisation im Gehirn," from *Ergebnisse der Physiologie*," Wiesbaden, 1902 and 1904. We have not been able to consider his latest communication in the *Ergebnisse*, vol. vi. See also the review by Hitzig, *Physiol. und klin. Unters. über das Gehirn, Ges. Abh.*, Berlin, 1904.

The individual segments of the brain surface are not of equal physiological importance: indeed, they differ extremely in this respect. We term each *cortical field* which controls a *definite function* a *centre*. The significance, site, and delimitation of these centres have been partly determined by experiment, and partly by the fundamental discoveries of Fritsch and Hitzig (*A. f. Anat. u. Physiol.*, 1870), followed by those of Ferrier, Goltz, Munk, Horsley, Sherrington, etc., but mainly by clinical observations and the corresponding post-mortem appearances.

It cannot be denied that Goltz's theory, at first so energetically opposed, has of late years gained more acceptance, as Munk, Hitzig, and in particular Monakow have become more reserved as to the view of sharply defined centres, and ascribe greater independence to the subcortical grey masses, whilst they do not regard the various functions as depending entirely upon circumscribed cortical areas. On the other hand a few writers, Mills [7] in particular, go decidedly too far in their acceptance and delimitation of circumscribed centres, as in their diagrams of localisation they represent as established facts much that is still hypothetical.

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[1] "Leitungsbahnen im Gehirn und Rückenmark des Menschen," 1876; also "Entwickl. d. Assoz. in menschl. Genhirn," *N. C.*, 1894; *N. C.*, 1898, 1899, 1901-3.

[2] *A. f. P.*, 1899.

[3] *Z. f. Hypnot.*, 1901; "Etude sur la myélinisation," Paris, 1900; and "Arbeiten aus d. neurobiol. Inst.," Jena, 1904.

[4] *A. f. P.*, Bd. xxxix.

[5] "Studien über die Hirnrinde des Menschen," translated into German by Bresler, i.-v.

[6] *C. f. N.*, 1894.

[7] Univ. of Penn., 1904; *Med. News*, 1904.

The recent development of brain surgery has afforded many opportunities for applying electrical stimulation to the exposed human cortex and thus establishing important facts. Investigations of this kind have been made by Horsley, Keen, Oppenheim, Starr, Bechterew, Negro-Oliva, Leobet, Heaton, Stewart, F. Krause,[1] Mills, Sinkler, and others. Lamarq [2] had some time before collected the results of such investigations.

Mills and Frazier [3] give a comprehensive description of their own and other observations, and their paper, like Krause's, contains an account of the technique of electrical stimulation.

Motor Zone.—The motor zone comprises the area of the motor centres. These extend over the *central convolutions* and the *paracentral lobes* (see Figs. 266, 267, and 268 ; also 255). According to previous investigations the anterior central convolution was thought to be the chief motor zone,

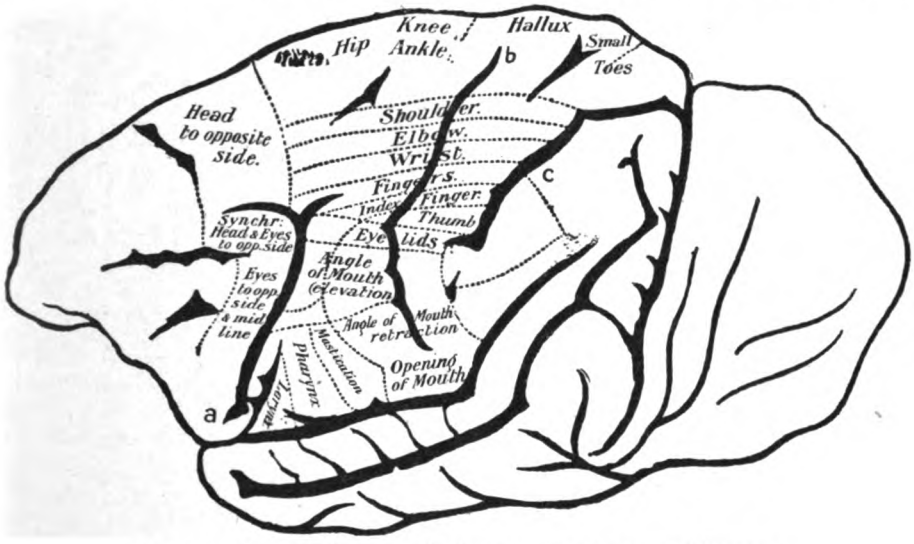

FIG. 266.—Motor region of cerebral cortex after Beevor and Horsley.

but Sherrington [4] has recently maintained, from experiments which he has carried out along with Grünbaum upon the brain of the ape, that the motor zone at the convexity includes only the anterior central convolution and the fissure of Rolando (Fig. 267), no foci of stimulation being found on the posterior central convolution. This view had already been advanced by Hitzig in 1874, and it has been accepted by Kocher, Monakow, Cushing, and others. It is also supported by the facts established by Vogt, Brodmann, and Campbell, as mentioned above. Vogt [5] gave me an opportunity of satisfying myself from the brain of the ape that these views were substantially correct.

Some cases of operation on the human brain have shown similar results, and Krause in particular from his own wide experience has confirmed the correctness of Sherrington's view. A few

[1] " Brain Surgery," in *Die Deutsche Klinik*, etc., 1904.
[2] *Arch. clin. de Bordeaux*, vi., 1897.
[3] Univ. of Penn. *Med. Bull.*, 1905.
[4] *Proc. Roy. Soc.*, vol. lxix., and " V. Kongr. f. Physiol.," Turin, 1901.
[5] See also the minute studies of C. and O. Vogt in the *Journ. f. Psych.*, viii.

writers, such as Rothmann (*A. f. Anat. u. Phys.*, 1907), Probst (*Sitz. d. Kais. Akad.*, Wien, 1906), and Gordon still, however, raise objections to it.

Brodmann (*Journ. f. Psychol.*, vi.) shows that the layer of the giant pyramidal cells,—the area giganto-pyramidalis,—lies within the zone of electrical stimulation, but is not absolutely identical with it. Flechsig (*N. C.*, 1903) finds a distinction between the anterior and posterior central convolutions in the distribution of the medullated fibres.

It is doubtful how far the motor area extends into the neighbouring segments of the frontal lobe. There is every indication that the most posterior area of the frontal convolutions in man belongs to the motor area.

The centres for the muscles of the opposite side of the body are so

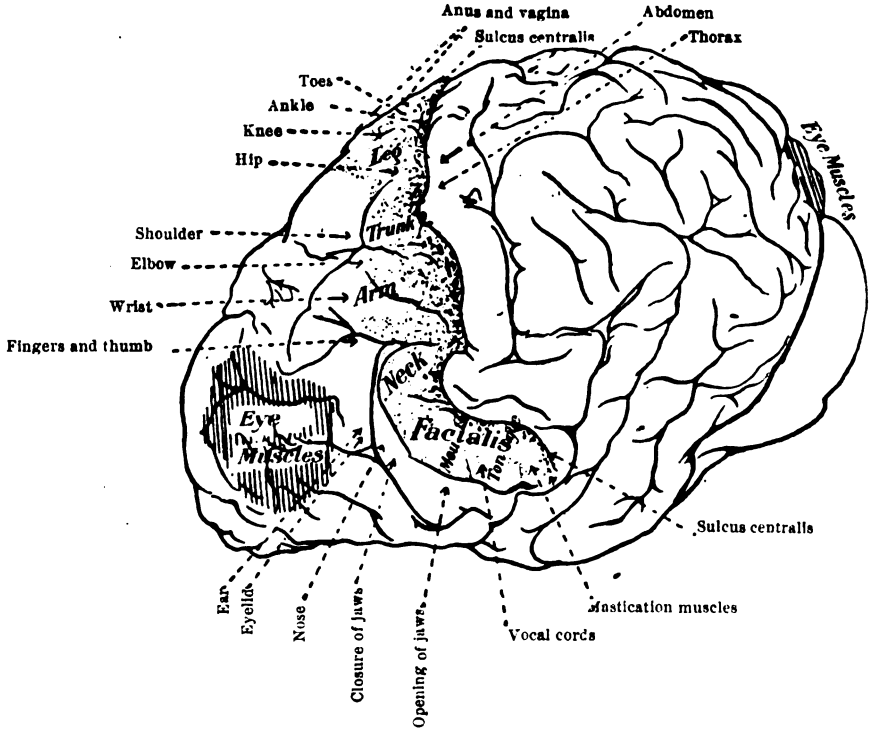

FIG. 267.—Lateral view of brain of chimpanzee with the motor zone and the special sites of representation for the various limbs, etc., after Grünbaum and Sherrington. The area of the motor zone is dotted black. It extends both into the central and precentral sucli. The arrows indicate the special field for each group of muscles or movements within the dotted zone. (After Monakow.)

distributed within the area in question that the centre for the *facial* and *hypoglossal* (probably also for the motor fifth) is situated in the lowest third or fourth of the anterior central convolution, whilst the *arm centre* occupies either mainly or exclusively the middle third or the middle two-fourths of the *anterior* central convolution and the central sulcus. The highest area of the motor zone is occupied by the *leg centre*, but it also extends to the paracentral lobes.

Although these centres are by no means definitely separated from each other, overlapping, as they do at their margins, and being partially identified with each other, further experience, particularly that gained

by modern brain surgery, has shown that a differentiation is possible within the individual centres, as there is apparently a special cortical area corresponding to each muscle group and each muscular function.

It is probable that in regard to the localisation of the motor centres a far-reaching analogy obtains between the human brain and that of the higher apes (Horsley, Sherrington), but it must be admitted that the details of the distribution are by no means established beyond all doubt. We think it, nevertheless, advisable, for the purpose of comparison, to present the facts established with regard to the brain of the ape (Figs. 266, 267).

Investigations in this direction show that we are dealing, not with separate centres for single muscles, but for simple movements.

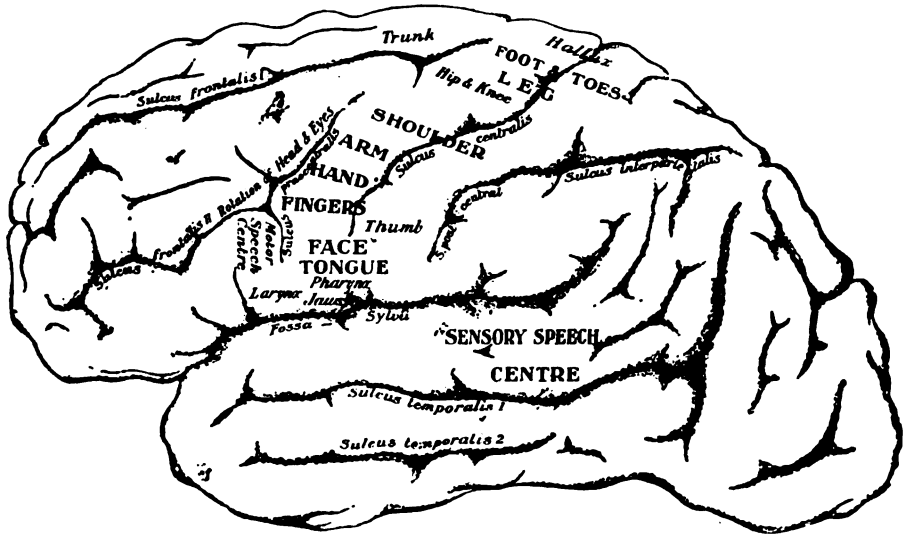

FIG. 268.—Motor region on the convexity of the human cerebral cortex. (The imperfectly determined localisation areas are distinguished by small print from those definitely established.)

We may regard it as established that in man also the zones for the thumbs and index-fingers lie lowest down within the arm centre (Fig. 268), that directly above these lie the centres for the other fingers and for the movements of the hand, the centre for the shoulder muscles occupying the highest area. This is a view which I must unconditionally maintain, both on account of numerous personal experiences—chiefly in dealing with focal diseases which have given rise to monoplegic symptoms, localised by operation or by autopsy—and of the results of electrical stimulation of the human cortex.

Mills and Frazier go still further in the mapping out of special centres, but we cannot here discuss their results.

As yet our knowledge of the distribution of the centres for the leg is not absolutely definite, and we do not know whether those for the muscles of the foot, toes, or thigh occupy the lowest point. The data seem to be very contradictory. It is not impossible that the localisation is in this

case less fine and more diffuse than in the arm region, as the movements of the lower limbs are much more of an elementary common character than the movements of the hand, which are finely specialised. I think it probable from my own experience, that the muscles supplied by the peroneal nerve have their centres at the convexity. In man also there seems to be a centre for the extensor longus hallucis, situated at the highest point of the central convolutions. The paracentral lobe undoubtedly plays an essential part in the cortical innervation of the lower limbs.

It would appear to be the general rule that the finer and more complicated the movements of which the muscles are capable, the larger are the centres which govern them (Starr, Obersteiner).

Numerous observations show that the centres for the muscles of the larynx, of deglutition and mastication, localised first by H. Krause,[1] then by Semon and Horsley,[2] Beevor,[3] Réthi, Ivanow, and others at the most anterior part of the foot of the anterior central convolution in animals, occupy the same site in man, although some doubts have been expressed on this point (Charcot-Pitres,[4] etc.).

There is still great difference of opinion as to the finer localisation of the motor centres in the foot of the central convolution, *i.e.* in the operculum. Some writers, such as Monakow, connect the posterior area of the third frontal convolution with the innervation of the tongue and partly also of the larynx, whilst Brissaud localises the laryngeal centre at the point where the horizontal joins the ascending branch of the Sylvian fissure.

Réthi (*Wien. med. Presse*, 1894) found a point on the cortex in the motor area, where stimulation produced a number of purposeful, orderly movements of mastication, deglutition, etc.

The *facial centre* is certainly situated in the lower fourth of the anterior central convolution. A very small area of this is regarded as the centre for the superior and inferior facial, but the question of the innervation of the ocular branch of the facial is not yet definitely settled (see below).

Some light is thrown upon the *importance of the motor zone* by the fact that its stimulation produces contractions in those muscles on the opposite side of the body which are under its control, whilst its destruction causes paralysis in the same muscles.

As we shall show later, strong stimulation of the motor region gives rise to contractions which pass in a regular manner from the groups of muscles first involved to others on the same side, and ultimately become generalised. Diffuse convulsions may thus be induced from this area of the brain.

According to recent investigations carried out specially by Sherrington and Hering (*Proc. Roy. Soc.*, 1897, and *A. f. d. g. Phys.*, Bd. lxviii.), the cortex of the motor zone sends out not merely impulses which produce contraction of the muscles, but also inhibitory impulses, which cause the contracted muscles or its antagonists to become relaxed.

It remains to be noted that some of these centres control not only the muscles of the *opposite* side, but also to a lesser degree the corresponding muscles of the same side of the body (Broadbent). This is the case as regards muscles which have as a rule not a unilateral, but a bilaterally symmetrical action : *e.g. the muscles of mastication, deglutition, of the larynx, the trunk, and those which close the eyes.*

[1] *A. f. Anat. u. Physiol.*, 1884. [2] *D. m. W.*, 1890, and *Practitioner*, 1899.
[3] *Phil. Trans. Lond.*, 1894. [4] *Arch. de Neurol.*, 1894, and *Arch. clin. de Bordeaux*, 1894.

Accordingly unilateral stimulation of these centres produces bilateral contractions, as H. Krause, for example, has demonstrated in regard to the tensors of the vocal cord, but a unilateral lesion is never the cause of a persistent paralysis. This would only result from a bilateral affection of these cortical centres, and would then involve both sides. Monakow suggests that the muscles mentioned which have a bilateral function are perhaps mainly supplied by subcortical centres and receive but little innervation from the cortex. The symptomatology of diseases due to bilateral affection of the motor zone is distinctly in favour of the correctness of Broadbent's theory.[1]

Cases pointing to cortical paralysis of the vocal cords in man (Dejerine, Rossbach, Eisenlohr,[2] Garel, Meillon,[3] Grasset) are few and uncertain. The importance of the laryngeal centre has also been called in question by Bechterew, Klemperer,[4] and especially by Onodi,[5] whilst on the other hand it is suggested (Masini, Brissaud,[6] Wallenberg [7]) that the corresponding cortical area controls mainly or even exclusively the adductors of the *opposite* side. The question must be regarded as still undetermined, although I think it likely that the suggested cortical centre for the larynx does exist in man and acts as a rule upon the muscles of both sides. I have been specially led to this view by some cases of diplegia, which commenced with almost complete aphonia. Katzenstein (*B. k. W.*, 1905) has recently published further experimental contributions to this question.

It is obviously not impossible that individual conditions may also play some part in localisation, that for example in some individuals the motor centres of one hemisphere may greatly predominate, so that a unilateral brain focus may produce paralysis of the whole tongue (glossoplegia), difficulty of deglutition, etc. I have observed a few cases which I could not interpret otherwise. This, however, is extremely rare.

As regards the muscles of the tongue, Monakow thinks that, in so far as they are concerned with the act of speech, they are innervated only by one hemisphere (the left), whilst the functions of deglutition, mastication, etc., are controlled by both hemispheres.

Centres for the movements of the eyes and the head, and in particular for the turning of the head and eyes towards the opposite side, have been found in the frontal lobes, mainly in the posterior region of the first and second frontal convolution (Ferrier,[8] Horsley,[9] Mott,[10] Bechterew [11]).

[1] We must not, however, take the idea of the motor centres to mean that all voluntary movements in their widest range are related to this area, and have here their only site of origin—their sole central representation. The majority of brain physiologists—Goltz, Schiff, Munk, Hitzig, and lately also Monakow—think rather that motor acts which are phylogenetically old, congenital, or acquired soon after birth, *e.g.* chewing, swallowing, walking, running, etc. (Munk's common or principal movements), have their origin, not in the cerebrum, but in subcortical motor centres. The cortex itself only exerts a starting and regulating influence on these movements. On the other hand the finely graduated muscular movements (Munk's particular movements) are entirely related to the motor zone. We should not, however, forget that these centres gain in importance and influence as we ascend the animal scale.

Monakow thinks that motor impulses originate also from other cortical areas, especially from the sensory centres. He does not admit that the psychical preparations of voluntary movement, the motor ideas, are associated with the motor region.

But, as Monakow shows, circumscribed cortical lesions give rise to symptoms of paralysis which are more severe than one would naturally expect according to this statement, because the groups of connected neurones which co-operate in order to carry out a common function, are injured as a whole by the loss of one of their constituent parts. The disturbance of function which is thus produced (Monakow's diaschisis) persists until paths are formed for new connections, new relations are established between the neurones and the functions are thus restored.

[2] *A. f. P.*, 1888. [3] *Thèse de Paris*, 1897. [4] *N. C.*, 1896.

[5] "Congr. med. int.," Rome, 1894; *B. k. W.*, 1894, and *A. f. Laryng.*, 1899.

[6] *Mal. de l'encéphale*, i. [7] *N. C.*, 1896.

[8] "Lectures on the Functions of the Brain." German by Weiss, 1892.

[9] Beevor and Horsley, *Phil. Trans. Lond.*, 1890; they found these centres in the middle part of the anterior central convolution and in the foot of the second frontal convolution.

[10] *Br.*, 1890. [11] *N. C.*, 1898.

This seems to be the case in man also, but unilateral affection of this area is never accompanied by corresponding symptoms of paralysis.

Sahli (*A. f. kl. M.*, Bd. lxxxvi.) has described a case which may be interpreted in this sense. A case published by Klien (*Z. f. N.*, xxvi.), which is given in support of this view, is not conclusive. See also Müller (*Z. f. N.*, xxii.), Klaas. "Über konjung. Augenablenkung," *Diss. Marburg*, 1898, zur Verth (*Mitt. aus Grenzgeb.*, xiv.), Sterling (*Arch. f. Anat. u. Phys.*, 1903).

The innervation of the trunk muscles is ascribed by Munk, Anton, Weber, and others to the frontal lobe, by Horsley, Schäfer (and Bruns), to the marginal convolution, and by Sherrington to an area between the centres for the arm and leg. The fact that other writers (Jackson) attribute to the cerebellum a special influence upon the trunk muscles does not exclude the possibility of a frontal cortical centre to which it is subordinate.

As regards innervation of the eye muscles, centres have been also found in other cortical regions, viz., the inferior parietal lobes (Ferrier,[1] Hitzig, Wernicke,[2] Monakow, Bernheimer), the occipital lobe (Ferrier, Schäfer, Munk, Obregia,[3] Berger,[4] Grünbaum, and Sherrington), the temporal lobes, etc. At all events a tract apparently arises from the visual sphere, which conducts impulses for the focussing of the eyes. In any case it is probable that the centre for *voluntary* movements of the eyes, etc., belongs only to the region indicated in the frontal lobe. Monakow thinks that motor acts which effect the movements necessary for the increase or for the suppression and prevention, etc., of the sensory stimulus concerned, are directly transmitted from any sensory sphere of the cerebral cortex (without the intervention of the motor zones). Mills agrees with him.

The relation of the angular gyrus to the superior levator palpebræ (Grasset and Landouzy) is absolutely uncertain. I have twice had an opportunity of applying electrical stimulation to this part of the brain in man, without eliciting any sign in the ocular muscles; in both cases, certainly, there was a tumour involving the medullary layer of the parietal lobe. There were, moreover, no symptoms of paralysis in the ocular muscles, and these did not even appear directly after removal of the cortex of the angular gyrus. The investigations which have more recently been made by Gerwer, Heaton, Roux, Silex, and du Bois-Reymond as to the cortical innervation of the eye muscles have not led to any uniform results.

Affection of the *optical orientation* had been noted by Sachs, specially by Exner, and also by Reinhardt, Zenner (*N. C.*, 1893), in disease of the inferior parietal lobe, and had been ascribed to the lesion of the association tracts between the optic centre and the cortical field of the eye movements in the angular gyrus. Pick and Anton (*W. kl. W.*, 1899) are also inclined to attribute to it affections of orientation and of deep localisation (and accommodation), corresponding to the experimental deductions of Munk, Schäfer, and Demoor, and to my own clinical observations. Bilateral destruction of the inferior parietal lobule causes loss of power to localise and to estimate distances (Anton). In one case of probable bilateral disease of the parietal lobes Hartmann found involvement of the stereoscopic vision (astereoscopia), marked affection of orientation, diminution of the power of perception, etc. He has written a monograph upon the subject of orientation (Leipzig, 1902), and Claparède has also studied it thoroughly (*Arch. de Physiol.*, 1903). Munk [5] has again recently described special relations between the angular gyrus and the ocular muscles and especially the eyeball; he localises his "ocular sensory sphere" in this area. This is disputed by Hitzig, Kalberlah (*A. f. P.*, Bd. xxxvii.), and others. Centres for the pupil of the opposite side have been found in the cortex by Ferrier, Bechterew, Pilcz, Parsons, and others.

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[1] "Localisation of Brain Diseases." German by Pierson.
[2] *A. f. P.*, xx.
[3] *A. f. Anat. u. Phys.*, 1890.
[4] *M. f. P.*, ix.
[5] "Über die Ausdehnung der Sinnesphären," etc., *Sitzungsber. d. K. Pr. Akad.*, 1901.

According to Bechterew [1] they are situated in the angular gyrus and the parieto-occipital fissure. A response to stimulation has, however, been elicited from other sites, such as the frontal lobe.

Vasomotor centres have been localised in the central convolutions and their neighbourhood, especially for the opposite half of the body. An influence upon the heart and vessels, upon the production of heat, and upon the vegetative nervous system has been attributed to these centres (Eulenburg - Landois,[2] Lépine,[3] Pitres, Franck, Bechterew, Ossipow, Parhon-Goldstein [4]), but as regards man at least, all these connections are still hypothetical, although vasomotor disorders in the opposite side have occasionally been observed after surgical operations upon this area (*e.g.* by H. Schlesinger and Oppenheim).

Lewandowsky and Weber [5] think they have proved experimentally that the vasomotor do not exactly coincide with the motor zones.

The same doubt applies also to the influence assumed on the ground of animal experiments by François-Franck, Bouchefontaine, Bechterew, Ostankow, Schukowski, and Beyermann, of the motor cortex upon the respiratory movements, to the so-called centres for the secretion of saliva and gastric juice (Lépine-Bouchefontaine, Bechterew and his school), and those for erection (Pussep, etc.), and for the secretion of sweat (Gribojedow).

The site of the cortical centres for the bladder and intestine in man is not yet known. Animal experiments have led to the view that they lie in the neighbourhood of the motor zones (F. Franck,[6] Bechterew,[7] Misslawsky and Ossipow, Frankl-Hochwart.[8] Frankl-Hochwart and Fröhlich (*N. C.*, 1904) succeeded in producing relaxation of the sphincter vesicæ from the cerebral cortex. Bladder affections are seldom observed in unilateral diseases of the cerebrum (Troje, etc.); they must undoubtedly occur in bilateral diseases under certain conditions, but we have few, if any, convincing cases of this kind. Czylharz and Marburg [9] have collected all so far reported; they localise this centre in man in the motor region between the arm and leg centres, or in the hip region. Friedmann (*M. m. W.*, 1903) explains one of his cases in this way. The subject is discussed also by Homburger (*Therap. d. Geg.*, 1903).

A few experiments (Quincke, Kirchhoff, etc.) led to the supposition that the cortex contains *trophic* centres for the muscles, etc., of the opposite side, but this is entirely hypothetical (see below). See the literature in Steinert (*A. f. kl. M.*, Bd. lxxxv.).

The Speech Centre.—In right-handed people the speech centre is connected with the *left* hemisphere, where it comprises the area of the *third frontal convolution* in its most posterior zone, and also, according to Wernicke, that of the *first temporal convolution*—especially the posterior two-thirds—and possibly also the angular gyrus. The posterior part of the third left frontal convolution contains the *motor* speech centre, the stations in which the idea is translated into words (see section on aphasia). The posterior zone of the right third frontal convolution appears to have a small share in the act of speech, whilst in left-handed persons it forms the main centre. The view that the corresponding area of the right frontal convolution constitutes a special centre of articulation (Kattwinkel, Polenoff, Struppler) is not founded on sufficient

[1] *A. f. Anat. u. Phys.*, 1899.
[2] *A. f. d. ges. Phys.*, 1898, and *V. A.*, Bd. lxviii.
[3] *Rev. de Méd.*, 1896.
[4] *Roumaine méd.*, 1899, and *R. n.*, 1902.
[5] *Med. Klinik.*, 1906.
[6] " Leçons sur les fonct. motor. du cerveau," Paris, 1887.
[7] *N. C.*, 1898.
[8] *Jahrb. f. Psych.*, 1902; *N. C.*, 1904; also Frankl-Hochwart and Zuckerkandl in Nothnagel's " Handbuch," xix.
[9] *Jahrb. f. Psych.*, 1901, and *W. kl. W.*, 1902.

grounds. The first left temporal convolution, or the transverse convolution, along with the adjacent part of the first temporal convolution, represents the *sensory* or sound-image centre of speech (Wernicke's speech centre), *i.e.* the site assigned to the memory for word-sounds.

As regards the conception recently put forward by Marie (*Semaine méd.*, 1906) in opposition to the prevailing theory of Broca's and Wernicke's centres, see the section on aphasia. Monakow's view will also be discussed there.

The sensory speech centre is situated, in left-handed persons, in the right temporal lobe, which even in right-handed persons has a certain share, varying in each individual, in the act of speech. According to Flechsig this centre extends to the transverse convolution of the temporal lobe which lies in the Sylvian fissure (the roots of the first temporal convolution). The share in reading which has been assigned to the angular gyrus will be discussed later. There is as a rule no special centre for writing—localised by Charcot and Pitres in the foot of the second frontal convolution—although even now the existence of such a centre is maintained by Schupfer,[1] Bastian, and others. Bastian[2] believes, not in a motor speech centre, but in the existence of four sensory centres: a glossokinæsthetic (which is identical with the motor), a cheirokinæsthetic, an acoustic, and an optic. We cannot further examine these theories, nor that of Stoïch.[3] It is still doubtful whether the left island of Reil takes part in the central processes of speech. It probably contains nerve tracts which connect the motor with the sensory speech centres. Flechsig says that the island appears to be a centre uniting all the motor and sensory fields of the cortex concerned in the process of speech. Touche[4] and others think it plays a part in this process.

The left hemisphere is functionally the more important of the two, not only on account of the part it plays in the matter of speech, but also of the lead which it takes in complicated motor actions, the left upper extremity being, in this respect, under the control of the left hemisphere (H. Liepmann). For details, see the section on symptomatology.

Our knowledge as to the site of the *sensory centres* in the brain cortex is much less definite than as regards the motor centres. Here also it is based mainly upon the results of experimental physiology, especially those of Munk,[5] Hitzig,[6] Goltz, and Ferrier. Opinions as to the interpretation of the symptoms produced by lesion and extirpation of certain cortical zones in animals are, however, by no means unanimous. Nor, in spite of abundant material for observation, has clinical and pathological evidence led to any definitely established results or facts which admit of universal application.

The majority of workers who have studied the question from the

[1] *Rif. med.*, 1903; Gardinier takes the same view (*Amer. Journ. Med. Sc.*, 1903).

[2] "Aphasia and other Disturbances of Speech." German translation by Urstein, Leipzig, 1902.

[3] *M. f. P.*, xiii.

[4] *Prog. méd.*, 1901.

[5] "Über die Funktionen der Grosshirnrinde," 1881; "Über die Fühlsphären," etc., *Sitz. d. k. pr. Ak. d. Wiss.*, 1892 and 1901; *Arch. f. Physiol.*, 1877-89. His latest contribution to this subject is that in the *Sitzungeber. d. k. Akad. d. Wiss.*, 1903. See literature here and in Monakow (*loc. cit.*).

[6] "Unters. über d. Gehirn," Berlin, 1874; *A. f. P.*, Bd. xxxiii. u. xxxvi., also "Alte und neue Unters. über das Gehirn," *A. f. P.*, xxxvii.

experimental or clinical aspects, are inclined to accept to a certain extent Munk's theory of the importance of the motor zone as a sensory sphere, to assign to it a great rôle in the originating of sensations (Hitzig, Monakow), or entirely to identify the sensory with the motor centre (Dejerine,[1] Long,[2] etc.).

Others deny any relation between the central region and sensibility (Ferrier, Schaefer, Charcot-Pitres,[3] Mills, Ferenczi [4]), whilst many eminent workers, especially of late years, think the sensory functions are related only to the posterior central convolution (see below). The correctness of this view is supported by the experiments of Sherrington and Grünbaum, Vogt and Brodmann, Krause, etc., and the histological investigations of Vogt-Brodmann and Campbell. A case by Birt (*Brit. Med. Journ.*, 1904) shows that there may be no paralysis whatever in a circumscribed lesion of the posterior central convolution. Even among the upholders of Munk's theory there is great diversity of opinion as to (1) the extension of the sensory centres beyond the motor region, (2) the share assigned to circumscribed centres in the whole process of conscious sensation, and (3) the relation of the various qualities of sensation to the different cortical zones.

Most of them agree—and this is the standpoint which I have for a long time occupied—that the sensory sphere extends beyond the motor region to the parietal lobe. Further, Ferrier, Schaefer, Horsley, Flechsig, Walton and Paul, Monakow, and to some extent Probst, include the gyrus fornicatus in the sensory sphere. Hitzig and others (Bouchaud) oppose this view. H. Hoppe (*Journ. Nerv. and Ment. Dis.*, 1904) ascribes to the sensory centres a territory extending far beyond the motor area. F. Müller agrees in this (Volkmann's "Samml. klin. Vortr.," N. 394, 395). It has also been asserted that only the sensations of touch and position are perceived in the region of the central convolutions, whilst other sensory perceptions, in particular that of pain, arise in the gyrus fornicatus. The *parietal lobe* had already been indicated by Nothnagel and Luciani as a special centre for the transmission of the sense of position. A few clinical cases (Vetter, Grasset, Monakow, Redlich, Bruns, Oppenheim,[5] Lemos, Durante, Mills,[6] Spiller [7]), and an experimental observation by Starr confirm this view. The question cannot, however, be regarded as definitely settled. Mills and Weisenburg (*Journ. Nerv. and Ment. Dis.*, 1906) maintain the complete separation of the motor and sensory areas in the cortex and the localisation of the latter in the posterior central convolution and in the parietal lobe. As to the relation of the parietal lobe to stereognosis, see further on.

Hitzig agrees with Goltz, that subcortical centres enter into action in the production of simple sensations (and their translation into movements), whilst he assigns the formation of sensory ideas to the motor zone; it is to be regarded, not as a sensory sphere in Munk's sense, but as a sphere of conscious sensations. Walton and Paul [8] distinguish between centres of simple sensation (for touch, pain, temperature, etc.), which they localise in the parietal lobe and gyrus fornicatus, and the centres, situated further forward in the central convolution, for the associative connection of the various stimuli with ideas, stereognostic perceptions, and consciousness of locality. This view is not in accordance with the facts.

The subject has lately been most exhaustively studied by Monakow. He believes that indefinite sensations and the perception of pain, perhaps also of simple pressure, etc., may be produced at the most different parts of the cortex and are not associated with any circumscribed centre. On the other hand he assigns to the sensory sphere as a circumscribed cortical territory the office of distinguishing qualities and of localisation, and also an essential part in stereognostic perception (which is not, however, wholly dependent upon this zone). The sensory sphere is not confined to the central convolutions, but includes also the parietal lobe (supramarginal gyrus and anterior parts of the superior and inferior parietal lobe). The sense of locality and the muscular sense are more strictly connected than the other qualities of sensation with the Rolandic region, the cortical zone of which extends to the parietal lobe, etc. Monakow maintains that the *posterior* portion alone, and not the whole area of the central convolutions, should be regarded as the region of the sensory centres.

[1] *R. n.*, 1893.
[2] "Les voies centrales de la sensibilité," Paris, 1899.
[3] *Arch. d. Neurol.*, 1894.
[4] *Pest. Med. Chir. Presse*, 1903.
[5] *Mitt. a. d. Grenzgeb.*, vi., 1900 (see older literature here), and *M. f. P.*, xviii.
[6] *Journ. Nerv. and Ment. Dis.*, 1900, and *Amer. Neurol. Assoc.*, 1901, also *Journ. Amer. Med. Assoc.*, 1902; *Univ. of Penn. Med.*, 1904.
[7] *Journ. Nerv. and Ment. Dis.*, 1906.
[8] *Journ. Nerv. and Ment Dis.*, 1901, and *Br.*, 1901.

Clinical and pathological observations show that sensory affections, in the form of symptoms of irritation and paralysis, may develop in lesions of the Rolandic region. These are not, however, necessarily present, and may be absent or slight although there is extensive paralysis of cortical origin. There may even be widespread focal disease of the *anterior* central convolution without any sensory symptoms (cases of Mills, Monakow, Oppenheim, etc.). On the other hand it is possible that in lesions of the cortex the sensory symptoms may be most prominent, or unaccompanied by any motor symptoms. This is a very rare occurrence, and is in all probability due to lesions of the posterior central convolution and the adjacent parietal region. Cases of this kind are described by Knapp, Oppenheim, Mills, Spiller, Redlich, Monakow, and others. Mills maintains that the sensory centres for the arms, legs, and face are separated and arranged in the same way as the motor centres, and that they are directly connected with the latter by means of association tracts.

Some of the experiments in stimulation which have recently been carried out upon the motor region of the human brain are of great interest. By means of these, paræsthesiæ were produced in the corresponding areas of the opposite side, the patient not being under an anæsthetic, and subsequent removal of the cortical centre was followed by anæsthesia in the same areas (Negro-Oliva, Bechterew, Krause). I have seen anæsthesia after such circumscribed cortical lesions in the anterior central convolution, but I take it that the condition is due to traumatic alterations in the neighbourhood, or to diaschisis in Monakow's sense of the word. According to Krause bathyanæsthesia is most persistent under such conditions.

An attempt has been made to solve this question by ascertaining into which zone of the cortex the sensory nerve tracts (see below) lead, or which cortical zone has to be destroyed in order to produce degeneration in the sensory tracts. Flechsig, Hösel, Tschermak and Probst have maintained that the central convolutions, the posterior in particular, represent this area, but Monakow objects to the interpretation of their observations. He himself had seen secondary degeneration of these tracts only as the result of extensive lesion of the parietal lobe and the posterior central convolution, but never in a lesion limited to the Rolandic region. Vogt came to similar conclusions.

The results of Sherrington's recent investigations already mentioned, according to which the motor zone is practically limited to the anterior central convolution and the central sulcus, are possibly calculated to solve all these contradictions and to make this matter clear.

Taking all these facts into account we may make the following deductions :—

The sensory centres occupy an area of the cerebral cortex which borders upon the motor zone, and perhaps partially coincides with it, but which chiefly comprises the *posterior central convolution* and the *parietal lobe.* Although coarse, undefined sensations of pain, etc., may possibly be perceived in other regions of the cortex, *localised sensation*, *i.e.* the *differentiation* and *qualification* of sensations, is related to this zone. It is also concerned in the discrimination of sensations and the recognition of the form and constitution of bodies (stereognostic perception).

We are not yet justified in localising the various qualities of sensation

in different portions of the cortex, but there is every probability that the sense of position is mainly associated with the parietal lobe, which would account for the important part it plays in stereognostic perception.

The sensory spheres receive stimuli practically only from the opposite side of the body. Brissaud's opposing view lacks any real support.

Visual Centre.—The experimental investigations which have sought to establish the relations of the cortex to the act of vision (Panizzi, Hitzig, Munk, Goltz, Loeb, Luciani, Tamburini, Bianchi, Bernheimer, etc.) have led, it is true, to no unanimous results. But from them, and from anatomical, embryological, clinical, and anatomo-pathological observations on man, among which those associated with the names of Monakow,[1] Henschen,[2] and Flechsig are worthy of special note, we may infer that Munk's theory that the *occipital lobe contains the visual centre* is correct. In man, indeed, it occupies mainly (or, according to Henschen, exclusively) the calcarine fissure and the cuneus. Opinions of investigators differ as to its further course (Monakow, Flechsig, Vialet,[3] Bolton, Brissaud, Bernheimer, Gallemaerts,[4] Niessl-Mayendorf,[5] Beevor and Collier,[6] Quensel,[7] Probst,[8] etc.). It possibly includes the fusiform and lingual gyri, perhaps also the first occipital convolution, but in a case of abscess which permeated almost the whole of the fusiform gyrus I found no visual affection of any kind.

Brodmann has attempted, as Bolton had already done, to use the histological structure, and the distribution of the cell layers, as a means of defining the limits of the visual area in the cortex. He has found that the area of his calcarine type forms a cone, the base of which rests on the occipital summit and from there occupies the calcarine fissure at the median surface of the cortex, extending on both sides somewhat on to the cuneus and lingual gyrus. Similar results were obtained by Campbell,[9] who regarded as the visual centre the region characterised by Gennari's lines and assigned another area in the occipital lobe for the psychical elaboration of optic impressions.

Mott has treated the question from the point of comparative anatomy (*Arch. of Neurol.*, 1907).

Destruction of the visual centre gives rise to contralateral bilateral hemianopsia (see below).

The other cortical territories of the occipital lobe—perhaps also the left angular gyrus—appear to play a part in the interpretation, the translation into mental form of the visual impressions, and to belong therefore to the visual sphere in the wider sense of the word. Monakow would include this area in his conception of the physiological visual sphere, which he extends not only to the central visual field, but also to the cortical zones which send out the optically produced motor impulses for the ocular muscles, etc. Wehrli entirely agrees with him. Many writers (Dejerine, Ferrier, Bastian, etc.) maintain that the optical memory field for the images of letters and words lies in the left angular gyrus. According to Wilbrand the cortex at the convexity of the occipital lobe is an optical memory field, with which the memory images of visual perceptions are connected (see mind-blindness). It is open to question

[1] *A. f. P.*, xvi., xxiii., xxiv., and *loc cit.*

[2] "Congrès internat. de Paris," 1900; also "Pathologie des Gehirns," Upsala, 1903, and *Semaine méd.*, 1903.

[3] "Les centres cérébraux de la vision," Paris, 1893.

[4] *Bull. de l'Acad., etc., de Belgique*, 1902.

[5] *A. f. P.*, Bd. xxxix. See the literature here.

[6] *Br.*, 1904.

[7] *M. f. P.*, xx.

[8] *Sitz. d. k. Ak. d. Wiss.*, Wien, 1906.

[9] "Histological Studies in the Localisation of Cerebral Functions," *Proc. Roy. Soc.*, 1904.

whether there are special areas or layers for the senses of space, light, and colour. Marchand (*Nouv. Icon.*, 1903) and others think not.

Affections of the optic nerve which persist for years may under certain conditions result in atrophy of the occipital lobe. Leonowa found atrophy of certain cell groups in the calcarine fissure in anophthalmia, and she related the optical functions to certain cell layers, as Monakow had already done. Hanke (*Obersteiner*, x.) reports a similar case. Berger (*M. f. P.*, vi.) has been unable to confirm these observations, but he has seen atrophy of all the cell elements of the cortex of the visual centre following artificial suppression of the visual act. From his latest publications Monakow seems to attach little importance to all these investigations. In adult man, atrophy of the eyeball produces no change in the optic tract or in the visual centre (Probst, *M. f. P.*, xvii.).

The most important points as to the relation of the visual centre to the optic nerve tracts will be noticed later.

Munk's theory, supported by Henschen, that there is a projection of the retina upon the occipital cortex, is opposed by Hitzig and others, and Monakow accepts it only to a certain extent. Wehrli (*Graefes Arch.*, Bd. lxii.) specially denies the projection of the retina on the cortex and the independent circumscribed cortical representation of the macula. Vogt recognises the anatomical projection, but draws no conclusions from it as regards function. We can only here refer to the important observations and investigations on this subject by Hun (*Amer. Journ. Med. Sc.*, 1887), Brissaud (*Nouv. Icon.*, xv.), Beevor and Collier (*Br.*, 1904). Hitzig [1] thinks the deeper subcortical centres play a prominent part in the act of vision, whilst on the ground of animal experiments he is very sceptical as regards Munk's cortical blindness.

The view that, in addition to the occipital, there is another visual centre in the angular gyrus, which is connected with the whole retina of the opposite eye in such a way that its destruction causes *blindness* of the opposite eye, must, in view of recent experiments, be unconditionally abandoned, although Seymour Sharkey (*Lancet*, 1897) has supported it.

Some workers think that the macula of either side is represented in both visual centres (Wilbrand,[2] Gowers, Knies [3]), as unilateral diseases of the occipital lobe do not usually affect the function of the macula. The connection would be maintained by the fibres of the corpus callosum (Heine). The site of direct vision, however, may remain intact in bilateral disease (Foerster,[4] Sachs,[5] Gaupp, O. Meyer,[6] Laqueur,[7] Schmidt and others). Other factors, such as a better vascular supply of this area, must therefore be concerned, as Foerster thinks. Laqueur and Schmidt found in their case that the posterior part of the floor of the calcarine fissure was uninjured, and they regard this portion as the centre of the macula lutea. Niessl v. Mayendorf (*A. f. P.*, Bd. xxxix.) also assumes an isolated representation of the macula in the optic radiation and cortex. Monakow thinks that the ganglion groups corresponding to the macula are very numerous in the subcortical centres, and he raises the question whether the macula may not perhaps be represented in the whole visual sphere. Bernheimer agrees with him, and thinks that so long as the macula contains fibres which are still capable of conduction, its function remains unaffected. Wehrli agrees with this view, whilst Lenz (*Inaug. Diss.*, Breslau, 1905) strongly opposes it.

Disorders of the power of orientation, of judging distances, etc., have also been observed in affections of the occipital (and angular) lobe. Touche [8] localises " topographical memory " in the fusiform gyrus.

Niessl v. Mayendorf regards interruption of the commissural fibres between the two visual spheres (*i.e.* those of the corpus callosum) as responsible for disorders of orientation. See also Becke (*Z. f. Aug.*, xi.) on the pathology of the occipital lobe.

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[1] " Alte und neue Unters. über das Gehirn," iv.; *A. f. P.*, Bd. xxxvii.
[2] *Beitr. z. Augenheilk.* (Festschr. Foerster), 1895.
[3] *Z. f. Biol.*, 1897.
[4] *A. f. Ophth.*, 1890.
[5] " Arb a. d. psych. Klinik, Breslau, 1890 and 1895.
[6] *M. f. P.*, viii.
[7] *V. A.*, Bd. clviii., and *V. A.*, clxxv. Further literature is collected by Niessl v. Mayendorf, *A. f. P.*, Bd., xxxix.
[8] *Presse méd.*, 1901.

With regard to the *olfactory centre* nothing is definitely proved. A few observations (Jackson and Beevor,[1] Pitt,[2] Stewart,[3] Oppenheim,[4] Siebert,[5] etc.) indicate that a centre of this kind is contained in the *hippocampal* or *uncinate gyrus*. Diseases of this region may cause hallucinations of smell, or homonymous or bilateral anosmia. These are, however, absent in some cases (Bouchaud, Bayerthal, Bartels). This function has also been assigned to the cornu Ammonis, but Ossipow[6] opposes this view.

Taste has been attributed to a centre in the anterior region of the gyrus fornicatus, but this is still uncertain.

The experimental results of Gorschkow (*Arb. aus Bechterews Klinik*, ii.; *Monit. neurol.*, 1902), which point to another region and to separate centres for taste and smell, cannot in the meantime be adduced as solving this question,

An attempt has lately been made to localise the sense of hunger and of thirst at the basal surface of the temporal lobe (Paget).

The *auditory centre* lies in the cortex of the temporal lobe, and indeed in its most superior convolutions. Dejerine and Sérieux[7] localise it in the anterior area, Strohmeyer[8] in the first temporal convolution. Flechsig thinks that the portion of the first temporal convolution concealed in the Sylvian fissure—the transverse convolution—mainly forms the central auditory field. Quensel[9] agrees with him. Mills also includes it in the auditory sphere. This seems to be proved by the anatomical investigations of Probst[10] and Vogt. Bechterew[11] has described a very fine differentiation of this centre.

We cannot here discuss these views, nor Campbell's recent attempt to differentiate further the auditory sphere, nor the cell types of this region described by Ramon y Cajal and Rosenberg (*M. f. P.*, xxiii.).

Kalischer's experiments (*Sitz. d. k. Akad. d. Wiss.*, 1907) show that a differentiation of tones may take place even in the subcortical centres in dogs.

Pathology shows that the deafness which sometimes affects the ear of the opposite side in diseases of the temporal lobe is of short duration. It is to be assumed that the centre of each hemisphere is connected with both auditory nerves, so that paralysis of one is rapidly made good by the compensating action of the other, whilst bilateral affections produce deafness (Wernicke-Friedländer,[12] Luciani-Sepilli,[13] Mills, Anton, Sérieux-Mignot,[14] Mott,[15] and others)

We do not exactly know the functions of the other cortical regions.

The processes of thought are probably associated with the *whole cortex*. To all appearance, however, the *frontal lobes* play a prominent part in the higher mental functions. This theory was first assumed on the ground of experimental observations, and it has received pathological confirmation. It is specially supported by experience of cases of frontal tumour, and chiefly by the fact occasionally noted (Bergmann,[16]

[1] *Brit. Med. Journ.*, 1888, and *Br.*, 1889.
[2] *Br.*, 1898.
[3] *Br.*, 1899.
[4] *Mitt. a. d. Grenzgeb.*, 1900.
[5] *M. f. P.*, vi.
[6] *A. f. Anat. u. Physiol.*, Suppl. 1900.
[7] *Compt. rend.*, 1897.
[8] *M. f. P.*, x., with bibliography.
[9] *M. f. P.*, Bd. xx., with bibliography.
[10] *A. f. P.*, Bd. xxxiv.
[11] *A. f. Anat. u. Phys.*, 1899, Suppl.
[12] *Fortschr. d. Med.*, 1883.
[13] "Funktionslok. auf der Grosshirnrinde," etc., German by Fraenkel, 1886.
[14] *Nouv. Icon.*, 1901.
[15] *Arch. of Neurol.*, 1907.
[16] *Arb. aus. d. chir. Klin.*, Berlin, 1902, and *B. k. W.*, 1901.

Oppenheim, Devic-Courmont,[1] Friedrich,[2] etc.), that excision of a tumour compressing the frontal lobe was followed by disappearance of the mental symptoms.

The question, however, is still an open one. It has lately been studied by Anton-Zingerle,[3] Zacher, Durante,[4] Schuster,[5] E. Müller,[6] Phelps,[7] Mills,[8] Consiglio,[9] Veraguth and Cloetta.[10] H. Munk in his latest publications and Monakow maintain a very reserved attitude, whilst Bolton (*Br.*, 1903) maintains firmly the relations of the mental processes to the cortex of the frontal lobe. Flechsig (see below) regards its frontal and especially its parieto-occipital association centre as the main site of intellectual activity. Mills and Wiesenburg (*Journ. Amer. Med. Assoc.*, 1906) maintain the special importance of the *left frontal lobe* as the centre of the higher mental functions. As regards other functions assigned to the frontal lobes, see below, and compare the section on symptomatology.

Flechsig's division of the cortical region into *sensory* and *association centres*, founded mainly on his investigations on the development of the medullary sheaths in the various territories of the brain, is not accepted by the majority of writers. Among the association centres he includes segments of the parietal, temporal, and frontal lobes, which are characterised by the fact that they receive no fibres from the corona radiata, nor nerve tracts from the periphery (sensory organs, spinal cord, etc.), but enter into connection with other areas of the cortex only by means of association fibres. Broadbent had previously expressed a similar view. This theory is opposed by Monakow,[11] Sachs, Siemerling,[12] Bianchi and Rutishauser,[13] and by Cécile and Otto Vogt[14] in particular, on the basis of exhaustive investigations. H. Munk is also entirely opposed to it. Flechsig[15] himself has recently greatly modified and limited his theory, and Ramon y Cajal, Hartmann,[16] and Tschermak[17] have developed theories very similar to Flechsig's. Thus Ramon y Cajal teaches that in addition to the bilateral centres of projection there are other attention or memory centres localised mainly on one side, and Hartmann assigns to the (left) frontal lobe the function of a higher association centre for the motor area.

Motor Tracts

The fibres coming from the *motor centres*, converging from all sides, push towards the interior of the brain and form a part of the corona radiata. After they have occupied a comparatively extensive area in the centrum semiovale, they become compressed in the interior of the brain into a very narrow space within the *internal capsule*. The medullated fibres of the *internal capsule* form an anterior and a posterior limb. As Figs. 269 to 271 show, the former lies between the

[1] *Rev. de Méd.*, 1897. [2] *Z. f. Chir.*, Bd. lxvii.
[3] Bau, Leistung, etc., d. menschl. Stirnhirns, *Festschr. d. Graz. Univ.*, 1901. Also Anton, *M. m. W.*, 1906.
[4] *Brit. Med. Journ.*, 1902. [5] "Psych. Stör. bei Hirntumoren," Stuttgart, 1902.
[6] "Stirnhirn und Psyche," *Z. f. P.*, lix., and *Z. f. N.*, xxi.
[7] *Amer. Journ. Med. Sc.*, 1902. [8] *Univ. of Penn. Med.*, 1904, and *Med. News*, 1904.
[9] *Il Morgagni*, 1905. [10] *Z. f. N.*, xxxii.
[11] "Über den gegenw. Stand.," etc., *Ergebn. d. Physiol.*, 1902 and 1904.
[12] *B. k. W.*, 1898. [13] *M. f. P.*, v.
[14] "Myélinisation des hémispheres cérébraux, *These*, Paris, 1900. *Z. f. Psychol.*, 1900, and *Z. f. Hypnot.*, 1901.
[15] *N. C.*, 1906. [16] *M. f. P.*, xxi.
[17] Nagel's "Handbuch d. Physiol.," iv.

caudate and the *lenticular nuclei*, the latter between the *optic thalamus* and the *lenticular nucleus*. The pyramidal tract passes through the anterior two-thirds of the posterior limb, and to all appearance in such a way that the fibres originating from the facial centre mostly run forwards close to the knee or within it, whilst behind them lie the fibres for the

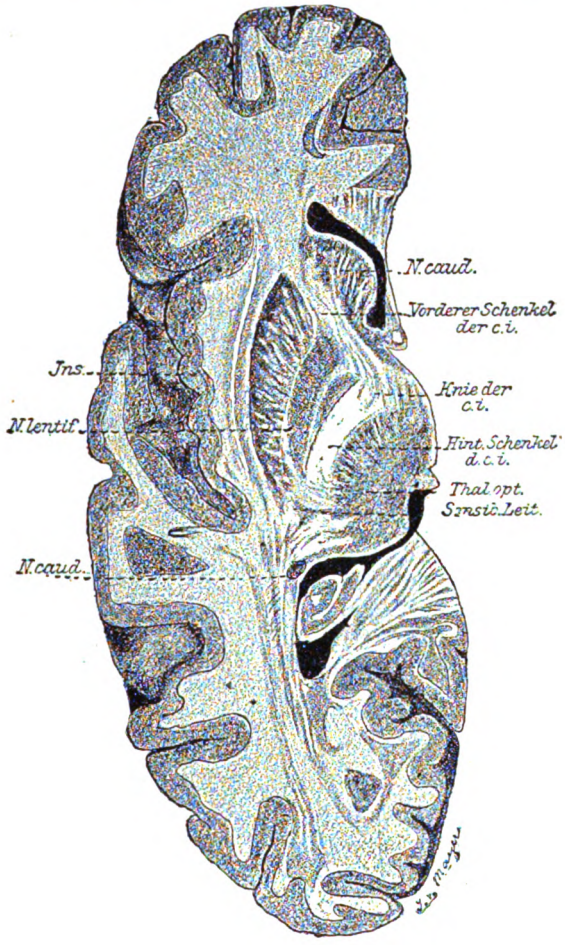

FIG. 269.—Horizontal section of human brain. (After Brissaud.) Vorderer Schenkel der c.i. = Anterior limb of internal capsule. Knie der c.i. = Knee of internal capsule. Hint. Schenkel = Posterior limb of internal capsule. Sensib. Leit. = Sensory path.

arm, and behind these again the fibres for the leg. The division is not, however, a sharp one.

A few bundles for the muscles of the tongue, jaws, and larynx have also been traced in the knee of the capsule, mainly by experiments on apes (Horsley and Beevor,[1] Semon). The tracts are apparently arranged one behind the other in the internal capsule, just as their centres of origin are arranged one above the other in the motor zone. The

[1] *Proc. Roy. Soc. Lond.*, 1890-91, and *Phil. Trans. Lond.*, 1890-91.

separation is not, however, by any means a sharp one, and the relations of position change at the different horizontal levels, so that morbid foci in the internal capsule usually affect all the motor fibres. It is also doubtful whether as regards finer localisation the facts established in animal brains may be applied to the brain of man.

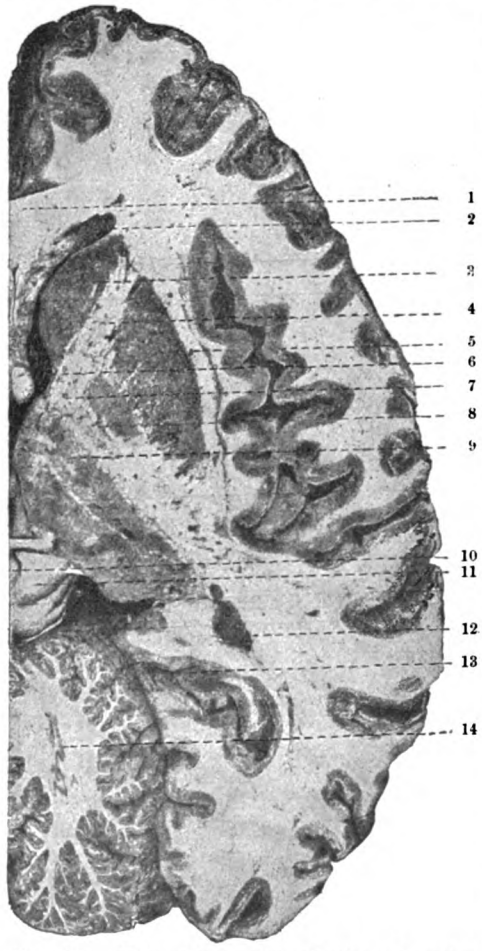

FIG. 270.—(After E. Flatau.) Horizontal section of human brain. From a photograph of a fresh specimen. 1. Corpus callosum. 2. Anterior cornu of the lateral ventricle. 3. Caudate nucleus. 4. Internal capsule (anterior limb). 5. Lenticular nucleus. 6. Pillar of the fornix. 7. Internal capsule (knee). 8. Internal capsule (posterior limb). 9. Optic thalamus. 10. Pineal gland. 11. Corpus quadrigeminum. 12. Inferior cornu of the lateral ventricle. 13. Parieto-occipital fissure. 14. Dentate nucleus of the cerebellum.

According to Monakow, the speech tract certainly passes through the knee of the internal capsule. The anterior limb contains the so-called fronto-pontine tract (see below).

French writers in particular (Abadie, Marie-Guillain, *Sem. méd.*, 1902; *R. n.*, 1902) deny that the fibre bundles are localised in the internal capsule in such a way that separate bundles can be distinguished for the face, the arm, the leg, the speech, and that there is a bundle containing only sensory fibres (see below). Dejerine has pronounced definitely against such a view. Personal clinical experience, however, has convinced me that the sensory tract occupies the posterior area of the posterior limb of the internal capsule, and that the bundles for the leg, arm, and face lie in this order in front of it. The division is of course not a sharp one.

From the internal capsule the pyramidal tract passes into the *pes pedunculi*, and there occupies an area more or less corresponding to that marked 2 and 3 (Fig. 273). The nerve tracts for the motor cranial nerves lie probably inwards from those for the extremities, whilst the median and lateral zones contain the fibres which arise from various areas of the cortex of the cerebrum, and which to all appearance terminate in the grey nuclei of the pons.

In the median bundle of the foot of the cerebral peduncle (see Fig. 273) there are fibres which arise from the frontal lobe (Flechsig,[1] Monakow, Anton-Zingerle)—or, according to Dejerine,[2] from the third frontal convolution and the foot of the central convolution—and pass to the anterior peduncle of the internal capsule in order to reach the pons. Some writers think some

[1] Flechsig has recently modified his views as to the origin of this bundle.
[2] "Anatomie des Centres nerveux."

of these fibres terminate in the optic thalamus (which does not accord with the experiments of Beevor and Horsley). Nothing is definitely known as to the significance of this tract. It has been thought that it served for the transmission of mental, affective impulses to deeper centres (Brissaud's psychical bundle). On the other hand it has been thought that it connects the cerebrum with the cerebellum by means of the grey masses which lie in the pons (see below). According to Dejerine's scheme, this bundle receives fibres from the foot of the peduncle which come from the speech centres and the cortical centres of the motor cranial nerves.

The relation of the foot of the cerebral peduncle to the fillet (see below) and the course of the fibres (cortico-pontine or cortico-bulbar) which run from the motor cortical centres to the corresponding nuclei of the pons and medulla oblongata are still very indefinite. Many writers have investigated this question (Flechsig,[1] Bechterew,[2] Edinger, Obersteiner, Hoche, Schlesinger, Probst, Muratoff, Weidenhammer, Romanoff, Hösel, Barnes, Collier and Buzzard, Boyce, Kosaka, Troschin, R. Sand [3]). Most of these writers think that this cortico-nuclear tract for the extremities runs in the foot of the cerebral peduncle, medial from the pyramidal tract. Others think that another fibre bundle runs in the lateral part of the peduncle, which passes into the so-called lateral pontine fillet or lemniscus bundle (Schlesinger). There is still some uncertainty as to the further course of these fibres within the pons. The view that they pass into the fillet and run downwards within it is opposed to the other view according to which they accompany the pyramidal tract and only intersect the fillet on their way to the nerve nuclei. Flechsig has given the name of " Fuss-schleife " to a special bundle which gradually passes from the foot of the cerebral peduncle into the median portion of the fillet. Spitska also describes bundles from the fillet to the peduncle, and Bechterew speaks of fibres of similar course as accessory fillet fibres. Probst (ref. *N. C.*, 1905) also mentions the " pyramidal fillet," which consists of the bundle from the peduncle to the fillet and of pyramidal fibres which lie dorsal to the crusta of the peduncle. We have no definite knowledge of the physiological significance of these fibres. They have been regarded on the one hand as a motor cranial nerve tract, on the other as a central, nucleo-cortical tract of the sensory cranial nerves. According to Sands's explanation, the cortico-nuclear tracts of the motor cranial nerves are blended with the pyramidal tract in the proximal segments of the pons, but leave it somewhat higher up, *i.e.* in a proximal direction from the corresponding nerve nuclei, in order to pass dorsalwards through the fillet and to join partly the contralateral, partly the homolateral nerve nucleus.

The lateral or Türck's bundle arises, according to Bechterew, Gerwer, and Hösel, from the temporal and occipital lobes, and according to Dejerine, Monakow, Kosaka, Archambault, and Probst, from the temporal only. The investigations of Marie and Guillain also support this view (*Semaine méd.*, 1903).

The foot of the cerebral peduncle is therefore composed of neurones or nerve-processes which have their cells of origin in the cerebral cortex.

Figs. 279, and 282 to 291, show the further course of the pyramidal tract through the *pons* and *medulla oblongata*. In the pons it is covered by the superficial layer of transverse fibres, and is divided by the intersecting pontine fibres into a number of bundles. In its course through the pons and oblongata it gives off at every level fibres which pass into the raphe, there become entirely or for the greater part crossed, and after decussation make their way into the *nucleus of the corresponding motor cranial nerve*. It has not yet been possible, however, to trace these tracts throughout their whole course. There is also, as already mentioned, a special bundle, which becomes independent in the lower segments of the pes pedunculi (or crusta), and which terminates below in the tegmentum—the " Fuss-schleife " or Edinger's cortico-bulbar tract, as the tract connecting the cortical centres with the motor nuclei of the cranial nerves is called. So far as observations go, the decussation takes place not far above, or on the cerebral side

[1] " Leitungsbahnen," etc., Leipzig, 1876.
[2] " Die Leitungsbahnen im Gehirn und Rückenmark," 2nd edition.
[3] *Obersteiner*, x. See bibliography here.

of the nucleus. The *main mass of the motor nerve tract* for the extremities, the cortico-spinal tract, passes down to the lower segments of the medulla oblongata, where *incomplete decussation* takes place (Fig. 274). The motor nerve tracts thus run from the centres to the spinal cord without being interrupted by grey matter. Details as to their position and distribution are given by Gattel, and in particular by Monakow. Pitres, Obersteiner, Probst, Rothmann, and Sand have also enlarged our knowledge of the course of the pyramidal tracts.

A small number of these motor tracts seems in man also to pass into the lateral column of the same side.

We have already referred to the individual differences in the condition of the pyramidal decussation. Absence of this pyramidal decussation is a very uncommon occurrence, but it has been found, *e.g.* by Pitres, and in a case of Zenner's. More recent experience and specially experiments on animals (Brown-Séquard, Wertheimer-Lepage, Starlinger, Hering, Gehuchten, Roth-

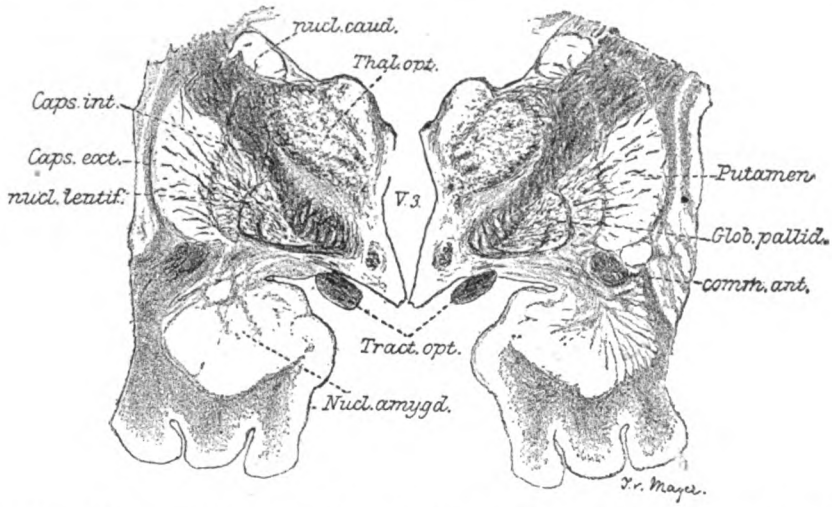

FIG. 271.—Part of frontal section of a brain through the internal capsule (stained by Pal's method).

mann, Prus, Collier, and Buzzard) point to the fact that motor impulses may also pass from the brain into the spinal cord by other paths. Monakow's bundle (the rubro-spinal tract) apparently represents one such second or accessory tract for motor conduction in the widest sense of the word. This is a fibre bundle which rises in the red nucleus, and soon after its emergence from this nucleus passes by way of Forel's tegmental decussation to the other side, and there runs downwards in the reticular substance of the pons, in order to pass into Deiter's nucleus (see below) and thence into the lateral column of the spinal cord (to pass downwards in the intermedio-lateral fasciculus (?), or in front of or within the pyramidal tract, Monakow, Held, Probst, Kohnstamm, Fraser, Lewandowsky, A. Thomas). For although it would appear from the published investigations at our disposal that this tract is of great importance for the conduction of motor impulses in the higher mammals, it is not yet clear whether it is also of essential importance in man. Monakow himself begs for greater care in the interpretation of the results of the study of secondary degeneration by Marchi's method. Marie and Guillain have not been able to isolate a corresponding definite bundle in man, whilst Collier and Buzzard (*Br.*, 1901) and Probst [1] (*Z. f. N.*, xv., with bibliography) are decidedly in favour of such a bundle. Monakow's bundle receives some fibres from the tegmental reticular formation (the lateral pontine tract of Tschermak, Lewandowsky). There

[1] Probst seems to have modified his views (see *Jahrb. f. P.*, xxiii.).

are other fibre bundles, which pass down into the spinal cord from the region of the corpora quadrigemina, the optic thalamus, the pons and Deiters' nucleus (according to the investigations of Haenel, Ernst (*Dissert.*, St. Petersburg, 1902), Gehuchten (*Névraxe*, 1904), Lewandowsky, and Bechterew), and which fulfil the function of conduction tracts for motor impulses (the extra-pyramidal tracts of Prus). Boyce, Russell, Kohnstamm, Wallenberg, and Probst have also studied this question. Rothmann [1] especially maintains the existence of extra-pyramidal motor conduction tracts, which he regards as of practical importance in man only in so far as they may undertake the rôle of a compensating tract after removal of the pyramidals. Probst has come also to this conclusion. Marie and Guillain (*R. n.*, 1904) interpret their results in favour of the existence of parapyramidal or extra-pyramidal tracts.

Lewandowsky was unable in his experiments on animals to find a direct path from the cortex across the thalamus to the red nucleus and Monakow's bundle, and he assumes a complicated circuitous path for the conduction by way of the cerebellum. He further refers to the descending tracts from the mid-brain, such as the tecto-spinal fasciculus, and to the descending fibre bundles to the spinal cord from the formatio reticularis of the medulla oblongata (reticulo-spinal tract, etc.). Bechterew (*N. C.*, 1906) on the other hand holds firmly to the view of a direct tract from the thalamus to the red nucleus, etc. Although we have no reason to assume that these tracts are concerned in the conduction of *voluntary* movements in man, yet they may play a part in the transmission of *affective* and *reflex* stimulations to the muscular system and also in the common movements of Munk (Probst).

Schütz (*N. C.*, 1902) describes a motor fibre bundle in the lateral part of the lemniscus (see below) which passes down from the cortical sensory centres into the spinal cord, but this requires confirmation.

The *facial tract*, which is situated in the internal capsule and probably directly beside the pyramidal tract in the foot of the cerebral peduncle, leaves the capsule at the anterior part of the pons, and after decussation in the raphe reaches the nucleus of the other side. Possibly a few of the fibres also make their way into the nucleus of the same side (Hoche, Kosaka, Sand). On the other hand some writers, as mentioned above, localise the cortico-nuclear facial tract in the so-called "Fuss-schleife." The fibres which arise in the *hypoglossal centre* pass over the lenticular nucleus and lie in the internal capsule between the tracts for the face and extremities. They become separated from the pyramidal tract within the pons (or in the cerebral peduncle ?), or in the medulla oblongata, and pass, probably medial from the lemniscus, backwards and upwards into the raphe, which they enter after decussation in the nucleus. A small number of the fibres seem to reach the nucleus of the same side. Sergi has made the most recent contribution to this question (*N. C.*, 1906).

We have little definite knowledge as to the course of the *motor speech tract*, but it evidently passes through the internal capsule (the knee) and the foot of the cerebral peduncle, where it occupies a position external to the hypoglossal tract (Raymond and Arthaud; Abadie, on the other hand, objects to the assumption of a special bundle passing from the speech centre through the internal capsule). Other bundles, such as the so-called "Fuss-schleife," Bechterew's accessory lemniscus, or Schlesinger's lateral pontine bundle (Mingazzini), have been regarded as the speech tract. Marie and others assume relations to the lenticular nucleus.

Our knowledge of the course of the *sensory nerve tracts* is particularly vague, but more and more light is now being thrown upon the subject.

The most recent bibliography will be found in a reference by Page May in *Br.*, 1906.

If we attempt first of all to trace these tracts on their way from the spinal cord to the brain, we should remember the fact, already mentioned on pp. 113-114, that some of them—those which ascend in the posterior columns—terminate in the so-called posterior column nucleus of the medulla oblongata, the nucleus gracilis, and nucleus cuneatus (Figs. 274, 275), whilst others, which have already decussated in the spinal cord, pass upwards within the antero-lateral columns. From the nuclei of the posterior columns fibres arise which make their way, in the form of internal arciform fibres, into the raphe and there decussate

[1] *N. C.*, 1902; *M. f. P.*, xii.; *Z. f. k. M.*, Bd. xlviii.; *M. f. P.*, xvi.

(Figs. 275, 276). This superior decussation, which is known as the *sensory decussation* or *decussation of the fillet*, undoubtedly contains a great number of the sensory nerve tracts, which then pass into the space between the olive and the raphe—the interolivary layer—and find their

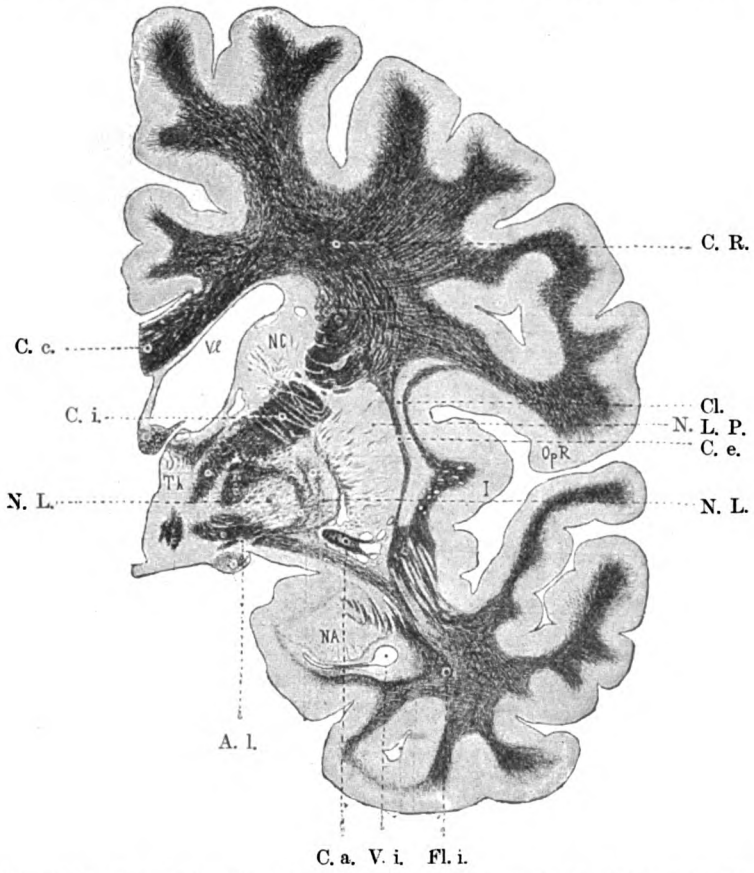

FIG. 272.—(After Dejerine). Frontal section through human brain. Stained by Weigert's method.

Op. R., Operculum. N. C., Caudate nucleus. V. l., Lateral ventricle. I., Island of Reil. N. A., Nucleus amygdalæ.

C. R., Corona radiata. Cl., Claustrum. N. L. P., Lenticular nucleus (Putamen). C. e., External capsule. N. L., Lenticular nucleus. Fl. i., Inferior longitudinal fasciculus. V. i., Inferior ventricle. C. a., Anterior commissure. II., Optic tract. A. l., Lenticular loop. N. L., Lenticular nucleus (inner part). C. i., Internal capsule. C. c., Corpus callosum.

way towards the brain as the *median* fillet or main fillet. It thus forms a sensory tract of the second order.

It is accompanied by the tract which ascends in the antero-lateral columns and which has already decussated in the spinal cord. According to some writers this mingles with the fillet fibres and occupies behind them a space between the olives, whilst according to others (Edinger,[1] Boyce,[2] Henneberg,[3] Horsley-Thiele[4]) it first passes into the ventro-

[1] *Vorl.*, etc., 1904.
[2] *Phil. Trans. Roy. Soc.*, 1897.
[3] *N. C.*, 1901.
[4] *Br.*, 1901.

lateral part of the reticular formation in the form of spino-thalamic and spino-tectale fibres. It is probable that these fibres serve chiefly for

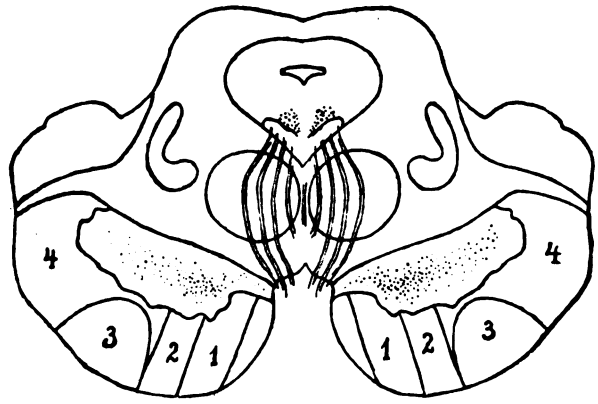

FIG. 273.—Diagram of tracts in the neutral portion of the crus cerebri (partly hypothetical). 1. Fibres from the frontal lobe to the pons. 2. Motor tract of the cranial nerves. 3. Motor tract of the extremities. 2. and 3. Pyramidal tract. 4. Fibres from the temporal and occipital lobes to the pons.

the conduction of the senses of pain and temperature, whilst the bundle in the superior pyramidal decussion probably conveys the impressions

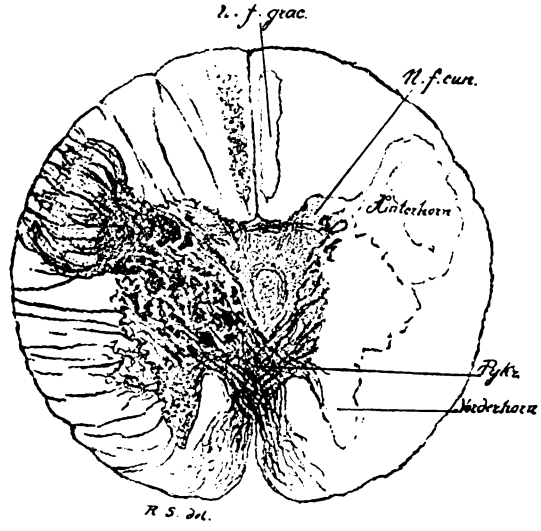

FIG. 274.—Section through the medulla oblongata at the level of the pyramidal decussation. (Stained by Weigert's method.) *N. f. grac.* = Nucleus funiculi gracilis. *N. f. cuns.* = Nucleus funiculi cuneati. *Hinterhorn* = Posterior cornu. *Vorderhorn* = Anterior cornu. *Py. kz.* = Pyramidal decussation.

made by the sensation of position and perhaps also by tactile sensations. In the proximal parts of the brain stem, however, these groups of fibres appear to become associated in the lemniscus (Henschen, etc.).

It appears very probable from the cases of Wernicke, Senator, Goldscheider, Bogatschow, Oppenheim, Wallenberg, Hun,[1] Breuer-Marburg,[2] and Rossolimo,[3] that the tracts in the oblongata which conduct the muscular sense or bathyæsthesia are distinct from the others and are contained in the parts of the interolivary layer contiguous to the raphe. As these bundles decussate only in the oblongata, their unilateral lesion may, according to its level, produce a crossed or homonymous disturbance of the sense of position (and motor ataxia ?). A unilateral focus may therefore give rise to *homolateral bathyanæsthesia* with *ataxia* and *contralateral analgesia* plus *thermoanæsthesia*. The tract for the tactile impulses is localised by Van Oordt in the ventro-medial part of the reticular formation. Wallenberg thinks it has a different course from those for pain and temperature. Dejerine and Long have pronounced against the view of separate fibre bundles for the conduction of the different kinds of sensation; but we must adhere to it. Babinski and Nageotte (*R. n.*, 1906) explain the symptoms by the assumption that the power of conduction differs for the various stimuli. Further contributions have been made to this subject by R. L. Müller (*A. f. kl. M.*, Bd. lxxxvi.), Henschen (*N. C.*, 1906), Babinski (*R. n.*, 1906), E. Mai (*A. f. P.*,

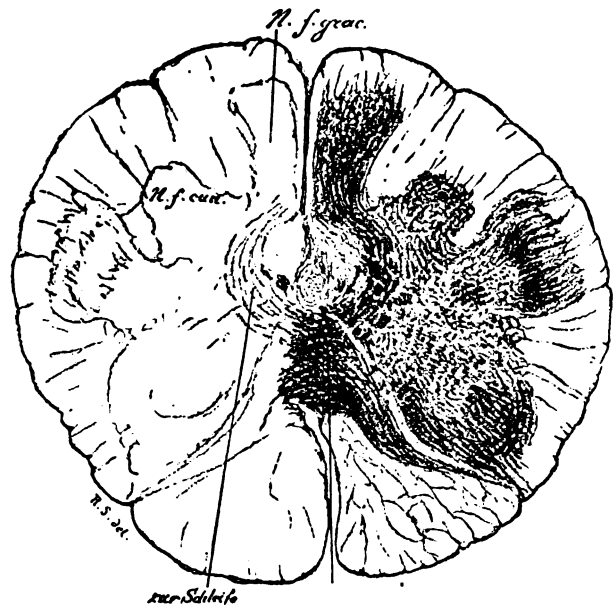

FIG. 275.—Section through the medulla oblongata at the level of the sensory decussation. (Stained by Weigert's method.) Schleife, fillet.

Bd., xxxviii.), Kutner-Kramer (*A. f. P.*, Bd. xlii.), E. Müller, and others. For the views of these writers see the introduction to the chapter on diseases of the pons, etc. Marchi's statement that fibres of the fillet originate in the cerebellum has not been confirmed by other writers.

Finally two other sensory tracts, viz., the *lateral cerebellar* and the *ascending antero-lateral* or *Gowers' tract*, ascend from the spinal cord. The former passes without decussation into the inferior cerebellar peduncle (restiform body) and makes its way through it to the cerebellum, and probably into the grey matter of the superior vermiform process. These fibres probably convey from the spinal cord, or from the periphery of the body, sensory impulses to the cerebellum which regulate the maintenance of equilibrium. Lesions of these fibre bundles seem to cause inco-ordination (cerebellar ataxia and homonymous motor ataxia).

As to Gowers' tract and its course see p. 112.

[1] *N. Y. Med. Journ.*, 1897. [2] *Obersteiner*, ix. [3] *Z. f. N.*, xxiii.

Kohnstamm thinks a considerable part of this tract reaches its provisional ending in the grey nuclear mass of the bulb or pons (senso-motor centre), and then passes up into the thalamus in the form of the bulbo-thalamic tract, whilst Lewandowsky adheres to the view that the tract terminates in the cerebellum. He assumes a second path for the sensory conduction above or through the cerebellum and the tract of the superior cerebellar peduncles to the cortical centre of the cerebrum.

From the posterior column nuclei fibres also pass by means of the so-called external posterior arcuate fibres into the restiform body and cerebellum (Hösel).

Bechterew ("Leitungsbahnen," and *N. C.*, 1897 and 1902), according to whose investigations a great number of the fibres which pass centralwards in the remains of the antero-lateral column are continued into the reticular formation, thinks some of these end in the grey nuclei of this region (central inferior and superior nucleus, reticular tegmental nuclei, etc.), and from there reach into the optic thalamus. Kölliker traces some of these tracts into the zone of the reticular formation lying dorsal from the intraolivary layer, and from these into the posterior longitudinal bundle, and

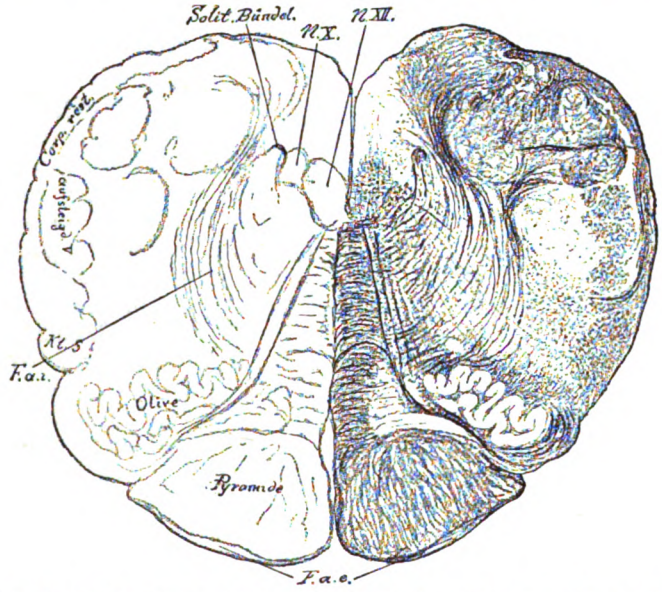

FIG. 276.—Frontal section through the medulla oblongata. *F. a. i.* = Internal arciform fibres. *F. a. e.* = External arciform fibres. (Stained by Pal's method.)

he suggests that sensory impulses may in this way be conveyed to the nuclei of the motor cranial nerves. The ganglion groups of the reticular formation are regarded by others (Gad-Marinesco, Kohnstamm) as respiratory centres. Kohnstamm calls them the co-ordination nuclei of the reticular formation and divides them into nuclei of the cervical reticular formation, of the medulla oblongata, and of the pons. Within the oblongata again he describes a lateral and a median nucleus; the former he associates with respiration and vasomotor functions. The salivary nuclei defined by Kohnstamm (*Anat. Anz.*, 1902) have already been mentioned.

There is also a bundle of fibres which passes from the cerebellum through the restiform body into the olive of the opposite side (see below).

Our knowledge of the fibre bundle of the reticular formation, described by Bechterew, viz., the *central tegmental tract*, which lies dorsal from the olives and ascends from them to the mid-brain (Figs. 285, 286), or takes a descending course and enters the olive (Thomas,[1] Marie-Guillain [2]), is as yet indefinite. Probst [3] calls it the olivary tract of the mid-brain, and thinks that it has the function of conveying the impulses of the mid-brain by means of the olive of the opposite side of the cerebellum. The connection between this fibre bundle and Helweg's triangular tract

[1] *R. n.*, 1903. [2] *Nouv. Icon.*, xvii. [3] *Jahrb. f. Psych.*, xxiii. and xxiv.

(Helweg-Bechterew bundle), assumed by Reinhold, Meyer, and by Thomas, are still hypothetical, as their existence has been inferred from cases of secondary degeneration which are not above objection (Obersteiner and others). Sorgo has denied these connections (*N. C.*, 1902). He traces the central bundle of the tegmentum from the olive to the superior cerebellar peduncles. As to the so-called tecto-bulbar tract, consult Kohnstamm (*W. C.*, 1903). We shall again revert to the tracts which connect the cerebellum with the oblongata, the cerebrum, and the spinal cord.

Investigations have yielded no consistent evidence as to the *further course of the main fillet* (median, superior fillet). The theory which Charcot specially advocates is that the sensory tract passes through the *posterior peduncle of the internal capsule*, and lies in its posterior third—in the retrolenticular part—behind the motor tract (carrefour sensitif).

Dejerine and his school maintain on the contrary that these fibres do not form a concrete bundle, but blend with the motor fibres, and that hemianæsthesia of the opposite side is produced, not by lesion of the "carrefour sensitif," wrongly so called, but of the thalamus (Dejerine Long,[1] Dejerine-Roussy,[2] Long-Dejerine [3]).

Schaffer is also opposed to Charcot's theory. We have, however, every reason from personal experience to adhere to Charcot's view, and we find further support for it in the results of Vogt's [4] investigations. Further, it can no longer be doubted that the sensory fibre bundles terminate wholly or for the greater part in the *central convolutions*, mainly or entirely in the *posterior* central convolutions and in the *parietal lobe*.

There are obvious differences of opinion with regard to the view of some writers (Flechsig, Hösel) that a great part of the lemniscus tract goes direct to the cortex. The majority (Monakow, Mahaim, Dejerine, Bechterew, Mingazzini, Ceni, Schlesinger, C. Mayer, Probst, Lewandowsky,[5] Münzer-Weiner,[6] Horsley, etc.) deny this and trace them first into the optic thalamus—and indeed into certain of its nuclei, especially the ventral—and from here to the cortex by means of a new neurone which has its origin at this point. This tract would therefore consist of at least three neurones: 1. the spino-bulbar, 2. the bulbo-thalamic, 3. the thalamo-cortical. It is possible that, as Monakow holds, other intercalary cells are interposed between them.

Various facts seem to indicate that some of the sensory tracts undergo interruption in the lenticular nucleus and in the still deeper grey masses, before they ascend to the cortex, but the relation of the lenticular nucleus or its loop (Fig. 272) to the fillet is denied by many writers (as well as Probst). Bechterew thinks that portions of the fillet extend into the subthalamic body, the globus pallidus, the reticular tegmental nucleus, and the optic thalamus. According to him only the so-called accessory fillet, which he regards as the supranuclear tract of the sensory cranial nerves, and the "Fuss-schleife," pass directly into the cortex. Gee and Tooth have assumed connections between a small number of the fibres of the fillet and the subthalamic body, etc. Possibly the fillet contains other short tracts, which interconnect the various levels of the pons and oblongata (Schlesinger).

According to Lewandowsky the fibres of the fillet end partly in the parageniculate nucleus between the median geniculate body and the posterior brachial quadrig., but the main mass terminates in the ventral nucleus of the thalamus!

A bundle of fibres, known as the tegmental radiation, which intersects the internal capsule, passes into the posterior central convolution and into the parietal lobe, and thence to the central

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[1] *Comp. rend.*, 1898; also Long, "Les voies centrales de la Sensibilité générale," Paris, 1899.
[2] *R. n.*, 1906.
[3] *R. n.*, 1906.
[4] "Verhandl. d. Anat. Gesellsch.," Rostock, 1906.
[5] *Journ. f. Psych.*, ii., and "Leitungsbahnen, etc.
[6] *M. f. P.*, xii., Ergänz.

ganglia and the subthalamic region, is included by some authors among the sensory tracts, but this view is disputed. Monakow especially opposes it, and he gives the name of tegmental radiation to other fibres which have nothing to do with the fillet.

We have therefore the following main paths for sensory conduction: the posterior column—posterior column nuclei—superior pyramidal decussation and internal arcuate fibres—intraolivary layer—main fillet passing to the cortex, either directly, or, as we can no longer doubt since the investigations of Monakow and Probst, after interruption in the optic thalamus. Then we have a second path which ascends in the antero-lateral column of the spinal cord, reaches the reticular formation of the brain stem, and then apparently joins or lies adjacent to the fillet at its higher levels. This path also reaches the optic thalamus and from thence passes to the regions of the cortex already named.

The sensory paths appear to become separated into different bundles for the various qualities of sensation at the point where the posterior peduncle crosses the internal capsule. Kirschhoff has attempted to define the tract for conduction of the pain sense. In one case of gunshot wound of the brain, in which everything pointed to a lesion of the carrefour sensitif, I found an isolated thermo-anæsthesia in one part of the opposite side of the body. Dejerine and Long deny this separation.

From the sensory cranial nerves and their nuclei there arises also a secondary conduction tract, which decussates in the raphe and makes its way to the cerebral cortex (Edinger, Bechterew, Schlesinger, Wallenberg, Probst, Hösel, Troschin, Hatschek [1]). It also appears to terminate first in the optic thalamus (Wallenberg, Kohnstamm). We have, however, no exact knowledge of the course of these central tracts of the sensory cranial nerves in man. They possibly join the main fillet, or go partly into the reticular formation.

Wallenberg has shown (*Anat. Anz.*, 1895, 1896, 1900; *A. f. P.*, xxxiv.; *Z. f. N.*, xxvii.) that after its emergence from the spinal root nucleus which accompanies the central tract, the trigeminus passes to the opposite side and then runs in the dorso-median zone of the medulla oblongata, below the hypoglossal nucleus. In the pons it separates, according to Hösel and Wallenberg, into two bundles, which first pass ventral to the abducens nucleus and at a higher level into the lateral region of the tegmentum of the pons, whilst Spitzer (*Jahrb. f. Ps.*, xviii.) thinks they go into the neigbourhood of the fillet. In his recent works Wallenberg traces this fibre bundle partly into the motor nuclei (iii. and v.) and partly along with the fillet to the optic thalamus and the "centre médian," but these facts, which were demonstrated chiefly on the brain of rabbits, cannot without further evidence be applied to the brain of man.

We should point also to similar investigations and statements by Ramón y Cajal, Gehuchten (*Névraxe*, 1902) Lewandowsky, Oordt, and Kalmus-Spitzer (*Obersteiner*, ix.). The latter writers think the trigeminal tract enters the main fillet in the form of the pontine fillet.

We cannot further discuss other unexplained tracts, such as the "tractus Probsti," and those which Kohnstamm traces out of the nucleus intratrigeminalis tecti. We must also pass over the pontine-lateral column tract (Tschermak, Probst, Collier, and Buzzard), the origin of which Lewandowsky traces to the nucleus reticularis tegmenti, and Kohnstamm to the nucleus reticularis magnicellul. pontis, and which then passes downwards in the area of the rubrospinal tract.

From all that has been said one fact seems certain, namely, that various paths are open to the sensory stimuli, and it is therefore conceivable that after one main tract is cut off they may be transmitted by accessory tracts. Further, the sensory tracts enter into connection with the motor nuclei of the cranial nerves and with the central ganglia, and form the paths by which reflex effects may be produced.

[1] *Obersteiner*, ix.

For the anatomy and physiology of the cerebellum a later chapter should be consulted. We are here concerned only with the tracts which connect the cerebrum with the spinal cord. They are contained in the cerebellar peduncles.

The inferior peduncle or the *restiform body* contains the direct cerebellar tract, along with fibres which pass from the posterior columns into the cerebellum, a tract—the cerebello-olivary tract—which connects the cerebellum with the olive of the opposite side, and another which brings nerve nuclei of the medulla oblongata (vestibular nucleus, Deiters' or Bechterew's nucleus) into relation with the cerebellum. Some writers however, do not include the corresponding fibre bundles with the restiform body.

The superior cerebellar peduncle, or tegmento-cerebellar tract, arises from the cerebellum, mainly from the corpus dentatum, undergoes complete or incomplete decussation below the corpora-quadrigemina, and then passes into the red nucleus. The latter is again connected by fibres with the optic thalamus and the cortex of the central parietal region. According to recent investigations, many of these fibres pass through the nucleus ruber in order to terminate directly in the optic thalamus (Monakow, Bischoff, Thomas, C. Mayer, Probst,[1] Mott, and Tredgold). The superior cerebellar peduncle therefore represents practically a connection of the cerebellum with the optic thalamus and through it with the cortex, but it is also a path by which stimuli may pass into the red nucleus and the optic thalamus and thence centrifugally to the spinal cord.

Lewandowsky traces an ascending branch from the superior cerebellar peduncle to the red nucleus, and a descending branch to the nucleus reticularis tegmenti.

The *middle cerebellar peduncle* consists practically of fibres which connect the cerebellum with the large nuclei of the pons of the opposite side (Ramón y Cajal, Bechterew, Mingazzini, Probst). These nuclei are joined by other fibres, which arise from the frontal lobe of the cerebrum, pass downwards into the median area of the foot of the cerebral peduncle, and with their terminal branches surround the grey nuclei of the pons. It is assumed that this frontal tract of the cerebrum and pons forms a connective system with the middle cerebellar peduncle of the opposite side, which establishes a relation between the cerebrum (frontal lobe) and the cerebellar hemisphere of the opposite side. Some writers, such as Bruns and Anton, have thus suggested that the frontal lobe is a centre subordinate to the cerebellum, a centre for the voluntary movements which maintain equilibrium.

Our knowledge of all these tracts, and of their physiological significance, is, however, incomplete and defective.

The following conceptions seem to us to be best founded :—

The inferior cerebellar peduncle contains chiefly fibres which convey to the cerebellum the impulses which effect and regulate co-ordination. These are unconscious stimuli penetrating from the periphery into the cerebellum and affecting its mechanism. This function is assigned to the lateral cerebellar tract, perhaps also to Gowers' tract or a part of it, and also to the ascending tracts in the posterior columns, which reach the cerebellum by means of the posterior column nucleus and the

[1] *A. f. P.*, xxxv.

arciform fibres arising from it. These spino-cerebellar tracts terminate in the cortex of the vermiform process. From the labyrinth also, whose importance as regards the maintenance of equilibrium can no longer be doubted, stimuli make their way to the cerebellum through the vestibular nerve and through a tract which originates in this nerve or its end nucleus and passes to the cerebellum.

The significance of the crossed cerebello-olivary tract is still quite uncertain (Henschen,[1] Thomas,[2] Bechterew, Babinski-Nageotte,[3] and others). Kölliker thinks it arises from the cerebellum and conveys impulses for co-ordinating muscular functions to the periphery. Bechterew includes the olives among the centres of equilibrium and thinks stimuli reach the cerebellum by way of the cerebello-olivary tract. In his opinion (supported apparently by investigations of Thomas and Marie-Guillain) the olives are connected with the *central bundle of the tegmentum* of the same side, by means of which stimuli are conveyed downwards from the grey masses in the region of the third ventricle. Probst also (*Jahrb. f. Psych.*, xxiii. and xxiv.) thinks the tract arises from the olives and makes its way to the cerebellum. Gehuchten agrees with him (*Névraxe*, 1904). Another view is that the cerebello-olivary tract forms a fibre system continuous with the superior cerebellar peduncle. Ceni has again recently upheld this view, as Sorgo has done (*N. C.*, 1902). According to Babinski and Nageotte these fibres terminate chiefly in the embolus cerebelli.

As to the superior cerebellar peduncle, it is not absolutely certain whether it conveys impulses to or from the cerebellum, or contains fibres with both these functions. It is probably mainly or exclusively a cerebello-fugal tract. It is suggested that the cerebellum may, by means of these fibre bundles, take part in the function of the motor cortical centres and thus exercise its co-ordinating power. Lewandowsky claims the tract as his second sensory nerve tract through the cerebellum. It is specially motor impulses, however, which follow this path from the cerebellum to the red nucleus, and from there to the spinal cord through Monakow's tract, etc. As regards secondary degeneration in focal diseases of the red nucleus, see also Marie-Guillain (*Nouv. Icon.*, xiii.).

Kölliker, Bechterew, and others describe tracts from the cerebellum, which leave it by means of the middle peduncle, and after interruption in the grey nuclei of the pons and oblongata, pass downwards into the spinal cord, where they attain the spinal muscular nuclei and thus directly influence the co-ordination of the muscles. Similar fibre bundles are contained in the reticular formation (and in the posterior longitudinal bundle), and lower down they pass into the region of the antero-lateral columns of the spinal cord. Fibres of this kind are also blended with the pyramids. Although a *direct* transition of the tracts from the cerebellum into the spinal cord is assumed by some writers (Marchi, Biedl, Thomas), it is denied by the majority (Russell, Kohnstamm,[4] Probst, Thiele, Lewandowsky, etc.), and it is now generally recognised that there are fibre bundles which extend first from the cerebellum into Deiters' nucleus, and after interruption there, pass down into the antero-lateral columns of the spinal cord—either without decussation or with a small proportion of the fibres passing to the opposite side (Orestano) of the spinal cord. These fibre bundles probably originate in the roof-nucleus, and pass within the fibres which Edinger has named the direct sensory cerebellar tract, to the nucleus of Deiters.

We must here remember that the nucleus of Deiters forms a point of junction for various tracts. In it terminates the vestibular nerve, and from it rises a tract which goes towards the cerebellum and thus forms a connection between the vestibular nerve and the cerebellum. It also constitutes a station of interruption for the fibre bundles which pass down from the red nucleus into the antero-lateral column of the spinal cord (Monakow's bundle). There is ground also for the view that it sends out fibres which pass in the posterior longitudinal bundle in the direction of the brain to the oculo-motor nuclei. The place of the labyrinth in the maintenance of the equilibrium, its influence upon the oculo-motor system, the ways by which the cerebellum may interfere in the mechanism of the movements of the head, trunk, extremities, and probably also of the ocular muscles will be explained when these anatomical relations are established. The subject has been thoroughly studied by Gehuchten (*Névraxe*, 1904 and 1906) and Lewandowsky (*loc. cit.*). As the superior cerebellar peduncles represent a crossed tract, and as Monakow's bundle again undergoes

[1] *Pathol. d. Gehirns*, i. [2] "Cerevelet," 1897. [3] *R. n.*, 1902. [4] *N. C.*, 1903.

decussation, we have thus a path by which the cerebellum may influence the same side of the body. Edinger believes in cerebello-nuclear or nucleo-cerebellar fibres, *i.e.* tracts which connect the cerebellum with the nerve nuclei, for all or most of the cranial nerves. Kohnstamm (*N. C.*, 1905) confirms this as regards the sensory trigeminus, while he defines a trigemino-cerebellar tract analogous to the lateral cerebellar tract.

The function of the middle cerebellar peduncle is as yet by no means fully explained. The theory has been put forward, and most clearly developed by L. Bruns, that the frontal lobe, as the highest voluntary centre for the trunk muscles, allows impulses to pass down through it to the cerebellum, the organ which unconsciously regulates co-ordination and maintains equilibrium, impulses which regulate its activity by voluntary influences. We shall return to this point, and to other questions upon the anatomy and physiology of the cerebellum in the section devoted to cerebellar diseases. Here we shall merely refer to the works of Bruce (*Brit. Med. Journ.*, 1899), Bruns (*B. k. W.*, 1900), Prus (*Poln. A. f. biol. med. Wiss.*, xxiv.), Lewandowsky (*Du Bois A.*, 1903), Pagano (*Riv. di Pat.*, 1902, and *Arch. ital. de Biol.*, 1905), Kohnstamm (*Pflüg. A.*, Bd. lxxxix.), Bolk ("Over de phys. beteek. van het Cerebell.," Harlem, 1903), Rynberk (*A. d. Fis.*, 1904), Clarke-Horsley (*Br.*, 1905), Horsley (*Brit. Med. Journ.*, 1907), Munk (*Sitz. d. K. Pr. Ak. d. W.*, 1906), and as regards the histology of the cerebellum to the paper by Bielschowsky-Wolff (*Journ. f. Psychol.*, v.).

The connection of the cerebellum with the cerebrum seems to be entirely a crossed one (Russell, Luciani, Wersiloff, etc.).

The *lateral fillet*, which arises at the level of the superior olive (Fig. 283), is probably a *sensory nerve tract of the second order, which receives acoustic impressions specially from the crossed auditory nucleus of the medulla oblongata*, and which probably conveys them through the *posterior corpora quadrigemina* or its posterior arm, and through the *internal geniculate body* to the *temporal lobe* (Monakow, Bechterew, Held, Baginsky, Ferrier-Turner, Obersteiner, Gehuchten,[1] Dantchakoff,[2] Lewandowsky,[3] Mahaim,[4] Quensel,[5] etc.). Ferrier and Turner deny that the posterior corpus quadrigeminum is in itself essential to the function of hearing. They regard the cochlear as the real auditory nerve. It terminates in the ventral auditory nucleus of the oblongata and in the tuberculum acusticum. From these nuclei, and from the former in particular, arise the trapezoid fibres (corpus trapezoides), which pass to the superior olive of the same and especially of the opposite side. Another neurone of the auditory tract takes its rise here, viz., the lateral (superior) fillet, which terminates partly in the posterior corpus quadrigeminum, in the lateral nucleus of the fillet, and in the internal geniculate body, whilst it again is connected with the cerebral cortex by a bundle of fibres running to the first temporal convolution. The auditory tract thus consists of at least four neurones.

Monakow thinks that the internal geniculate body has the function of an acoustic centre, but he points out that this is the part of the brain which degenerates after destruction of the temporal lobe. Tschermak thinks the auditory tract enters into connection with the optic thalamus (in the cat). Ramon y Cajal apparently holds the same opinion. He distinguishes between an *acoustic reflex system*, which terminates in the corpora quadrigemina, especially in the posterior one, and a *central auditory tract*, which makes its way through the peduncle of the posterior corpus quadrigeminum and the internal geniculate body, and after interruption in the optic thalamus ends in the cortex of the temporal lobe. Van Gehuchten speaks of two secondary auditory tracts, the ventral, which passes from the accessory nucleus into the corpus trapezoides,

[1] *Névraxe*, 1903 and 1907.
[2] *Bull. de l'Acad. Belg.*, 1902.
[3] "Unters. über die Leitungsbahnen," etc., Jena, 1904.
[4] *Cery*, 1905; *N. C.*, 1906.
[5] *M. f. P.*, xx. See also Winkler's work: "The Central Course of the Nervus Octavus," etc., Amsterdam, 1907.

and the dorsal, which goes as an independent bundle to the raphe, crosses it, and makes its way in the lateral fillet to the posterior corpus quadrigeminum. Lewandowsky's diagram of the primary and secondary auditory tracts is practically similar to that presented here. The fibres of the vestibular branch terminate, as he states, in the dorsal acoustic nucleus and in Bechterew's nucleus, and partly in the grey matter of Roller's auditory root. He denies direct but admits indirect connections with Deiters' nucleus. The central path of the vestibular nerve is doubtful. It perhaps passes through the olive and the olivo-corebellar tract to the cerebellum. Probst has shown that Deiters' nucleus has no connection with the function of hearing.

The Optic Nerve and Tract

The optic nerve undergoes partial decussation in the chiasma. Now that Kölliker has withdrawn his opposition, this fact is universally admitted. The great median bundle passes to the opposite side, whilst the lateral bundle remains uncrossed. See the diagram, Fig. 277. The optic tract of one side is therefore connected with the homonymous external half of the retina of the same side and with the internal half of the opposite eye.

The bundle supplying the macula lies apparently in the central part of the optic nerve, a position which it also occupies in the chiasma and optic tract.

The fibres of the tract end in the *pulvinar* of the optic thalamus, in the *lateral geniculate body*, and in the *anterior corpus quadrigeminum*. The lateral geniculate body forms the main terminal station of the optic fibres. It is improbable that the fibres for vision enter the pulvinar and the anterior corpora quadrigemina. It is much more likely (Monakow, Henschen) that the nerve fibres which pass into them serve to transmit the reflex movements, especially, as Monakow suggests, the pupillary light reflex (see further on).

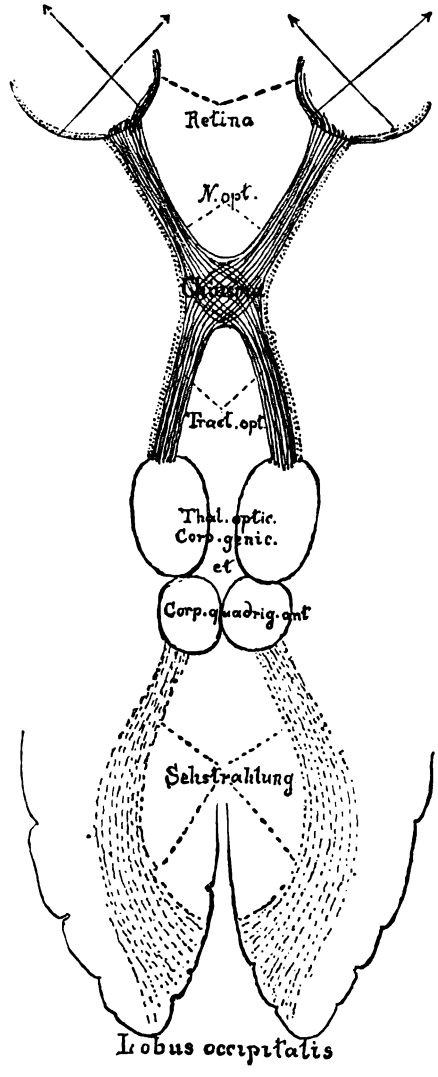

FIG. 277.—Diagramatic representation of the optic nerve tract (without regard to the proportion of the various parts, etc.). Sehstrahlung = Optic radiation of Gratiolet.

Bechterew has shown experimentally that destruction of the anterior corpora quadrigemina causes blindness. This is contradicted by the experimental results of Ferrier and Turner (*Br.*, 1901), and observations on man have proved that diseases of this segment of the brain need not

produce any severe affection of vision. This has been more recently emphasised by Bach [1] and by Uhthoff.[2]

The *end stations*, the *primary optic centres*, are connected with the *cortex of the occipital lobe*, the true optic centre, by long longitudinal tracts, which pass through the white matter of the temporal and occipital lobes. Many of these fibres may be traced into the cortex of the cuneus. The optic radiation passes along the outer side of the posterior horn. According to Henschen it traverses the temporal lobe at the level of the second convolution, and also passes through the centrum ovale of the inferior parietal lobe.

The optic tract was formerly localised in the so-called radiation of Gratiolet, but recent investigations, amongst which I would mention those of Flechsig, Probst,[3] Niessl, Mayendorf, Hösel, Redlich,[4] Lassalle-Archambault,[5] and Quensel,[6] leave no doubt that it makes its way chiefly through the region of the inferior longitudinal fasciculus, and that this latter does not form, as was previously thought, a system of association fibres between the occipital and the temporal lobes, but is in its main mass a centripetal system of projection fibres leading from the external geniculate body to the cuneus. Some writers assume, however, that it also contains bundles of association fibres (Förster,[7] Redlich, Archambault, and Tsuchida [8]).

Monakow thinks the optic radiation finds its way to the cuneus without passing through the inferior parietal lobe. According to his investigations (*N. C.*, 1904) other fibre bundles, which extend to the inferior parietal lobe, and especially to the angular gyrus and the occipital convolution, come from the pulvinar and the central nucleus of the optic thalamus.

The fibres of the optic nerve arise for the most part from the ganglion cells of the retina, and their terminal branches become lost in the primary optic centres. The second neurone arises from their ganglion cells, especially from those of the lateral geniculate body, and extends into the cortex of the occipital lobe. The optic radiation, however, also receives fibres which originate from the cortex of the occipital lobe and go to the primary optic centres (Monakow, Ferrier-Turner, Probst). Probst and Quensel think they pass through the optic radiation of Gratiolet. Nerve processes also go from the cells of the latter to the retina. Monakow believes that intercalary cells are inserted between the first and second neurone of the visual path.

It is generally admitted that other fibres arise from the occipital lobe and pass to the cells of the nuclei of the ocular nerves. Bernheimer thinks this conduction is carried on by association tracts between the occipital and angular lobes. According to Monakow, fibre bundles from the pulvinar and optic thalamus penetrate the parietal lobe, and on the other hand tracts pass from these areas to the primary optic centres.

Monakow found atrophy of the lateral geniculate body, the pulvinar, and the anterior corpus quadrigeminum after lesions in the cortex of the calcarine fissure. Leonowa also found atrophy of these structures in anophthalmia. Similar results have been described by Henschen, Moeli, Cramer, Zinn, Wickel, Bischoff, Probst, Bernheimer, Obersteiner, Spiller, etc. The process of degeneration has been traced as far as the corpus mamillare (Monakow, Marie-Ferrand). There is no doubt, however, that, in adults, foci may exist for a long time in the occipital lobe without producing secondary degeneration in these regions.

Brissaud found secondary degenerations in the tapetum of both sides, and also in the optic radiation of Gratiolet (*Nouv. Icon.*, xv.), in softening of the calcarine fissure.

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[1] *Z. f. Aug.*, viii.

[2] Graefe-Saemisch, "Handbuch," 2nd ed., T. 2. Consult also Herzog (*Z. f. N.*, xxx.) and Mörchen (*Z. f. Aug.*, 1903).

[3] *Sitzungsb. d. k. Akad.*, Wien, 1903 and 1906.

[4] *Obersteiner*, vii.

[5] *Nouv. Icon.*, xix. He names this fibre bundle the central optic bundle or the tractus geniculo-calcarin.

[6] *M. f. P.*, xx. See bibliography here.

[7] *A. f. P.*, xxxix.

[8] *A. f. P.*, Bd. xlii.

In addition to the visual path a number of other fibre bundles which are regarded as association tracts, have been traced in the occipital lobe (Sachs,[1] Forel, Monakow, Dejerine, Onufrowicz, Muratoff, Anton and Zingerle, Vialet, etc.). These include an occipito-frontal fasciculus for the connection of the occipital with the frontal lobes, the inferior longitudinal fasciculus (Fig. 278) for connecting the occipital with the temporal lobe, and the transverse fasciculus is supposed to connect the optic sphere and the speech centre—a view which, as already stated, can no longer be maintained.

The relations of the so-called occipito-frontal fasciculus have been disputed by Obersteiner and Redlich, who call it the reticulated cortico-caudal bundle, and also by P. Schröder, Probst, Hart-

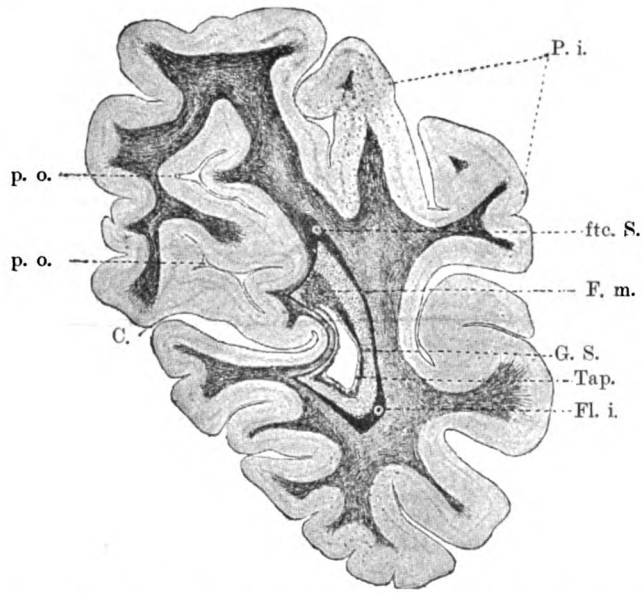

FIG. 278.—(After Dejerine.) Frontal section of posterior part of the præcuneus through the parieto-occipital and calcarine fissures. Stained by Weigert's method. P. i. = Inferior parietal lobule. ftc. S. = Fasciculus transversus cunei (Sachs). F. m. = Forceps major. G. S. = Optic radiation of Gratiolet. Fl. i. = Inferior longitudinal fasciculus. C. = Calcarine fissure. p. o. = Parieto-occipital fissure. Tap. = Tapetum.

mann, Arndt, and Sclarek. As regards the so-called tapetum (see below) some of these writers have arrived at conclusions which differ from those hitherto accepted. According to O. Vogt, the so-called occipito-frontal fasciculus contains at all events the longest known association fibres, but the connection of the occipital with the frontal lobes implied in the name cannot be substantiated. Quensel and Archambault also failed to demonstrate a fronto-occipital association bundle in man.

In the longitudinal fibres of the occipital lobe, *i.e.* the fibre masses surrounding the posterior horn, we may, from the investigations of Monakow, Dejerine, Gianelli, Brissaud, and others, distinguish three parts: 1. the inferior longitudinal fasciculus, or the stratum sagittale externum (Sachs); 2. the so-called optic radiation, or the stratum sagittale internum (Sachs); 3. the tapetum, or the fibres of the corpus callosum. The fibres belonging to the optic radiation, however, as already mentioned, run for the greatest part in the inferior longitudinal fasciculus. There are in

[1] "Arb. aus der Breslauer Klinik," 1890 and 1895.

addition commissural tracts which connect the two occipital lobes with each other, and the occipital lobe of the right side with the temporal lobe of the left. These all make their way through the splenium of the corpus callosum. These views, however, have not been definitely proved, either from the anatomical or the physiological point of view (Flechsig, Schroeder, etc.).

The Central Ganglia

Our knowledge of the significance of the *central ganglia* is still incomplete. The *caudate nucleus* was formerly thought to be connected with the movements of walking and running, but this view, though still held by a few writers, lacks confirmation. A *thermic* centre has also been localised in this region.

The *lenticular nucleus*, especially the globus pallidus, is regarded by some writers as a transmission station for some of the sensory nerve tracts, but this has been denied by others. *Motor* troubles—especially diminution of the power of articulation, of masticating and swallowing—have been observed in bilateral affections of this ganglion (Brissaud, Lépine, Mingazzini,[1] etc.), but it is doubtful whether these symptoms could be attributed to the lenticular nucleus itself. There must have been a lesion either of the neighbouring fibre bundles, or of centripetal tracts of the area of the lenticular nucleus, which are concerned in the reflex mechanism of swallowing, masticating, etc. At all events the cases to which we have access show that diseases of the lenticular nucleus and the caudate nucleus may run their course without producing any symptom of motor paralysis (Dejerine, Reichel). This is shown also by the experimental results of Probst, Stieda, Schüller, and others, with which Chaikewtisch's results do not agree. Marie (*Semaine méd.*, 1906, etc.) ascribes to the lenticular nucleus, to the left in particular, great importance in regard to articulation, as do Mills and Spiller (*Journ. Nerv. and Ment. Dis.*, 1907).

The *optic thalamus* has extensive relations with the cerebral cortex and the nerve tracts which ascend from the spinal cord. Monakow [2] in particular has demonstrated the connections between the various sections (nuclei) of the optic thalamus and the different regions of the cortex. The so-called peduncle of the optic thalamus establishes these connections. Moreover, the optic thalamus forms a transmission station, or an organ of reception for the stimuli which flow to the brain from the periphery. We have already referred to the relations of the optic nerve to this ganglion (pulvinar), to the cases which show that the sensory nerve tract, the fillet, undergoes a kind of transformation in the optic thalamus, or that it here acts upon groups of ganglia which only then transmit the impulses to the cerebral cortex, and to the relations of the superior cerebellar peduncle with the thalamus, etc. Monakow, with whom Probst [3] entirely, and Dejerine for the most part, agrees, states his conviction that all the sensory nerves enter into relation with the optic thalamus, before they reach the cortex. He suggests that each of these organs has a bilateral connection with the optic thalamus, so that lesion of one thalamus does not necessarily produce any definite symptoms of paralysis. He has not, however, sufficiently proved his theory, which is also held by F. Müller (Volkmann's "Samml. klin. Vortr.," 1905). We should nevertheless point out that other writers (Ferrier and Turner) have been led by their investigations to assume the existence of fibres connecting the thalamus to the opposite cortex, and that Probst describes fibre bundles passing from the fillet and the superior cerebellar peduncle into the optic thalamus of the opposite side.

Bechterew [4] regards the optic thalamus and corpora quadrigemina as reflex organs, which not only receive sensory conduction tracts, but also give rise to fibres which assume a centrifugal course and affect the muscular system. He reminds us of the descending tracts which arise from them, pass through the posterior longitudinal bundle, and thence into the antero-lateral columns of the spinal cord (see preceding section).

In spite of these results of anatomical investigation, some of which are still under dispute, we have but little positive knowledge as to the function of this part of the brain. It has not been proved, and is indeed improbable, that lesions of the pulvinar inevitably produce hemianopsia; this condition only results from injury of the external geniculate body of the same side. Nor has it been definitely shown that an affection limited to the optic thalamus gives rise to anæsthesia of the opposite side of the body, although, according to the prevailing views as to the course of the

[1] *Riv. Sper. di Fren.*, 1901-02. See also Franceschi, *Riv. di Patol.*, 1905.
[2] *A. f. P.*, Bd. xxvii. and xxxi., and *loc. cit.*
[3] *Jahrb. f. P.*, xxiii.
[4] "Westn. psych.," 1885; *N. C.*, 1897; and Ernst, *Dissert.*, Petersburg, 1902.

sensory nerve tracts we should expect this result, which, indeed, has been explicitly stated by Dejerine and his school (see below).

Many writers (Bechterew,[1] Nothnagel,[2] Brissaud) with whom Anton, Probst, Prus, Sternberg,[3] and others agree, are of opinion that the optic thalamus is a centre for *involuntary, automatic movements*, for the psycho-reflexes, and therefore for the movements which are not directly controlled by the will. Lesions of the thalamus, therefore, will, according as they have an irritating or a paralysing effect, produce either exaggeration or abolition of the automatic, mimic movements. Nothnagel shows that a disease of the optic thalamus may be revealed by a paralysis of the crossed facial which is only apparent during laughing, as the nerve is not under the influence of the will. According to Borst [4] the corresponding tract—the psycho-reflex facial tract—passes through the corona radiata of the optic thalamus and thence into the dorsal region of the pons. Kirschhoff [5] localises the mimic centre in the median nucleus of the optic thalamus, but Probst and Dejerine-Roussy disagree with this view. Involuntary movements (see section on hemiathetosis, hemichorea, etc.) have been specially observed in affections of the optic thalamus, and their interpretation has given rise to many theories. Are we, in fact, dealing with an irritation of a centre for involuntary movements? Or does the irritation of sensory fibres to the cortex cause stimulation of the motor regions which manifests itself in these involuntary movements? Are there fibres from the thalamus to the cortex for the inhibition of the functions of the motor region, whose interruption causes these symptoms of motor excitement, etc.? Or does the impulse for them arise, not from the optic thalamus, but rather from simultaneous lesions of an adjacent tract? We shall return to this question in the chapter upon focal symptoms.

Anton and Hartmann think that the optic thalamus excites automatic movements and the corpus striatum of the lenticular nucleus inhibits them.

Vasomotor functions have been attributed to the thalamus as well as to the corpus striatum (Schiff, Lusanna, Sinkler, Prus, Horsley, Bechterew-Ostankow), and also secretory and trophic functions (Marie, Probst). Parhon and Goldstein (*R. n.*, 1902) have collected all the available observations, and from their own experience and that of Schiff, Monakow, Horsley, White, and Marinesco, they conclude that the corpus striatum especially contains vasomotor centres. It also seems probable from cases of Hutchinson, Rezek, Engelhardt, Marburg,[6] Homburger, and Oppenheim, that in man also the central ganglia (corpus striatum, and the optic thalamus in particular) have an influence upon the emptying of the bladder. According to Réthi all the movements which precede the act of feeding may be originated by the optic thalamus. This is supported by observations which I have made in infantile pseudobulbar paralysis (*q.v.*) with regard to the "feed reflex."

The question of central tracts for mastication and deglutition has been studied experimentally by Economo (*Pflüg. A.*, Bd. xci.).

Homburger states (*N. C.*, 1903) that unilateral focal affections of the central ganglia produce transient incontinence of urine, persistent increase of the desire to micturate, and occasionally incontinence of fæces, whilst bilateral lesions result in persistent incontinence. The symptomatology of affections of the thalamus, which had already been considered in the monograph of Nothnagel, Oppenheim ("Hirngeschwülste"), etc., has of late years been specially studied by French authors (Dejerine,[7] Thomas-Chiray,[8] Dide-Durocher,[9] Dejerine, and Roussy.[10] Their conclusions will be discussed in the section upon symptomatology.

We have already alluded to, and will further discuss in the following section the relations of the *corpus quadrigeminum* to the ocular nerves, but these are still indefinite. Animal experiments have given conflicting results, as the discussion recently held between Prus and Bernheimer has shown. The symptomatology of affection of this region and of the corpus callosum, cerebral peduncle, etc., will be discussed elsewhere.

A centre for the formation of sounds (basal sound centre) is localised by Onodi and Bechterew in the region of the posterior corpora quadrigemina, and by Ivanow in the thalamus. Onodi has described a phonation centre in the area between the posterior corpora quadrigemina and the vagus

[1] *V. A.*, Bd. cx.; *N. C.*, 1903; *Rev. russ. de Psych.*, 1904; *M. f. P.*, xvii.
[2] "Top. Diagnostik der Gehirnkr.," 1879; *Z. f. k. M.*, 1889.
[3] *Z. f. N.*, xxiv. See also Berger, *Z. f. k. M.*, Bd. lii.; Reich, *N. C.*, 1904; Mann, *Brit. Med. Journ.*, 1905; Marburg, *W. kl. W.*, 1905.
[4] *N. C.*, 1901.
[5] *A. f. P.*, xxxv.
[6] *Jahrb. f. Psych.*, 1901; *W. kl. W.*, 1902.
[7] *R. n.*, 1903; *Gaz. des hôp.*, 1907.
[8] *R. n.*, 1904.
[9] *R. n.*, 1904.
[10] *R. n.*, 1906; Roussy, "La couche optique." Paris, 1907.

nucleus, whilst W. Sternberg and Latzko (*Z. f. N.*, xxiv.) have, from their observations on a case of anencephalus, assumed a deeper (bulbar) site for it. I think it doubtful, however, whether conclusions drawn from monsters should be applied to normally developed man.

Origin of the Cranial Nerves

The nuclei of the cranial nerves, which at the same time play the role of *trophic centres* for the motor nerves, lie in the grey matter which lines the floor of the third ventricle (in its hindmost area), the aqueduct of Sylvius, and especially the fourth ventricle, and partly also in the deeper layers of the pons and medulla oblongata.

The oculo-motor nerve (Fig. 279) has its origin in two long columns of nuclei, which lie in the *anterior corpora quadrigemina* below the aqueduct of Sylvius. It consists of a number of cell groups.

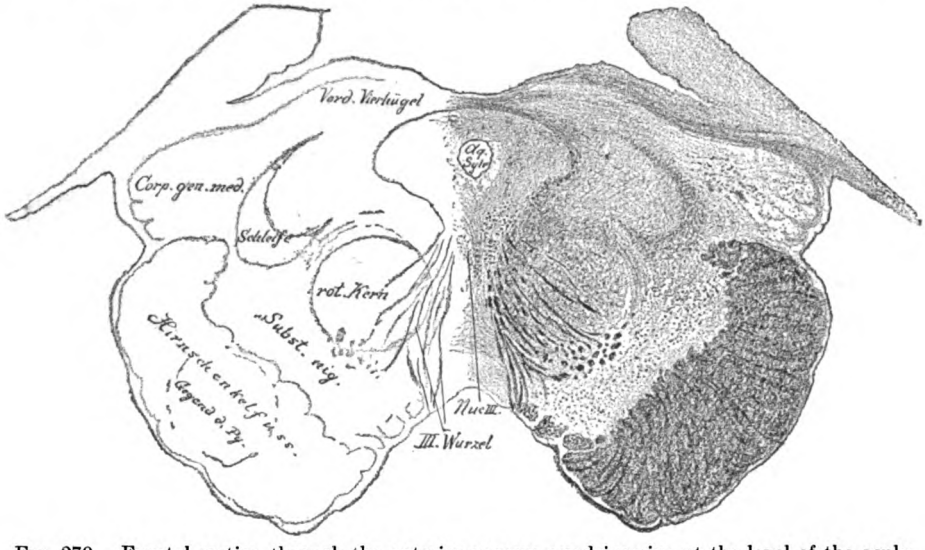

Fig. 279.—Frontal section through the anterior corpora quadrigemina at the level of the oculo-motor nucleus. *Vord. Vierhügel* = Anterior corpus quadrigeminum. *Corp. gen. med.* = Internal corpus geniculatum. *Schleife* = Fillet. *Rot. Kern* = Red nucleus. *Subst. nig.* = Substantia nigra. *Hirnschenkelfuss* = Crus cerebri. *Gegend d. Py.* = Site of Pyramid. *Nuc. III.* = Third nucleus. *III. Wurzel* = Root of third nerve.

Attempts have been made by many writers to define the nuclear groups of the oculo-motor nerve. Perlia speaks of a main group of the oculo-motor nucleus, which he again subdivides into four parts, anterior, posterior, dorsal, and ventral. There is also an unpaired central nucleus (Spitzka's sagittal nucleus) and the Westphal-Edinger groups in the anterior region of the nucleus. Then, most cerebralwards, where the Sylvian aqueduct opens into the third ventricle, comes the anterior nucleus of Darkschewitsch. Kölliker speaks of a main nucleus, which at its cerebral end gives off a round, dorsal nucleus. The main nucleus has two subdivisions: a dorso-lateral group with large cells, and a dorso-median with small cells. There is also an unpaired central nucleus in the median plane.

The diagram after Siemerling here reproduced (Fig. 280) shows the lateral large-cell nucleus as the main site of origin of the oculo-motor nerve, but the unpaired large-cell central nucleus is also included in the area of this nerve. The nucleus defined by Darkeschewitsch has probably nothing to do with the oculo-motor nerve (Cassirer-Schiff, Siemerling, Mpnakow, Majano.[1] It has lately

[1] *M.f. P.*, xiii.; see earlier bibliography here; consult also Wilbrand-Saenger, "Die Neurolgie des Auges," and Uhthoff, "Die Ophthalmoplegien"; Graefe-Saemisch, "Handbuch," 2nd ed., 1906. Panegrossi has studied the comparative anatomy of the oculo-motor nuclei (*M. f. P.*, xvi.).

been brought into relation with the posterior longitudinal bundle and regarded as its nucleus (Bernheimer, Probst, Panegrossi). It has been doubted by several writers whether the Westphal-Edinger groups belong to the oculo-motor nerve. A breaking-up of the large-cell lateral nucleus into subdivisions does not appear to be practicable.

It is probable that the various groups of nuclei form centres for the different muscles supplied by the oculo-motor nerve, but it has not hitherto been possible to apply these limitations to man in any convincing way, and among recent writers Bach in particular has opposed the view of special nuclei for the different eye muscles. Hensen and Voelkers, as the result of their experiments on dogs, localise in the most anterior position a nuclear area for the ciliary muscle and the sphincter iridis; behind it, the nucleus for the internal rectus muscle; then follow those for the superior rectus, the levator palpebræ superioris, the inferior rectus, and the inferior oblique. According

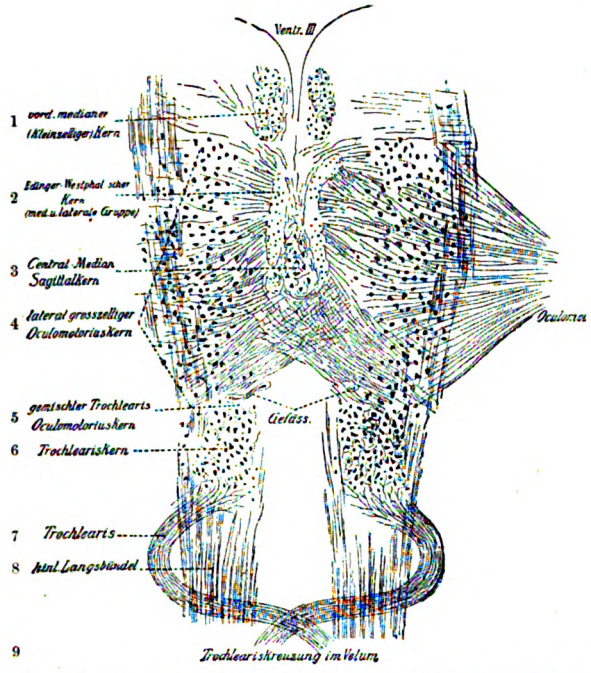

FIG. 280.—Grouping of the nuclei (after Siemerling.) Diagram of a horizontal section. 1. Anterior median small-celled nucleus. 2. Edinger-Westphal nucleus. Median and lateral groups. 3. Central median nucleus. 4. Lateral large-celled oculomotor nucleus. 5. Mixed nucleus of oculomotor and trochlearis (iv.). 6. Nucleus of trochlearis. 7. Trochlearis (iv.) nerve. 8. Posterior longitudinal fasciculus. 9. Decussation of trochlearis in velum. *Gefäss* = Arteries to nucleus.

to Kahler and Pick, the *pupillary fibres* are in man contained in the *most anterior root bundles*, and the fibres for the external ocular muscles in the most posterior root bundles, the median portion of those fibres being destined for the internal and inferior recti. Cases of Marburg,[1] Oyon, and others also support the view that the pupillary fibres lie in the most anterior root bundles. An old observation by Leube accords with this view. Bernheimer and Majano regard the cell groups of the lateral main nucleus as the nuclei of origin, and give them in the following order, from in front backwards: levat. palpebræ superioris, rectus superior, obliquus inferior, rect. int., rect. inferior. As to the nucleus of the levator, see also Dejerine-Gauckler-Roussy (*R. n.*, 1904).

Since the discovery of Nissl's staining method attempts have been made to determine by its means the nuclear groups for the various ocular muscles (Gehuchten, Bernheimer, Gehuchten-Biervliet, etc.). According to Bach and Marina, removal of the iris and the ciliary body causes

[1] *W. kl. W.*, 1905.

changes not in the region of the oculo-motor nucleus, but in the ciliary ganglion. Bach absolutely denies the relation of the Edinger-Westphal nucleus to the sphincter pupillæ (*C. f. N.*, 1906). This theory is strongly, but unconvincingly opposed by Bernheimer (*N. C.*, 1899). He still maintains that the anterior accessory nucleus of the oculo-motor nerve innervates the interior muscles of the eye, and he has produced immobility of the pupils in apes by destruction of these groups. Ruge (Graefe's *A.*, Bd. liv.) and Levinsohn have had similar results. The question, however, is not yet solved. Monakow thinks it not impossible that the groups of nuclei for the interior muscles of the eye are very scattered, only the corresponding root fibres being contained in the most anterior bundles. He also suggests that the internal rectus may have a double nuclear origin, one in the posterior area of the crossed dorsal nucleus, and another for the movement of convergence in the single central nucleus. Majano also admits this. From the observations which we have on man Monakow concludes that a lesion in the posterior segment of the red nucleus may involve the oculo-motor root bundles for the levat. palp. sup. and the rect. int. of the same side, etc.

We can only refer to the most recent work on the subject by Tsuchida ("Arb. aus Monakows Institut.," ii., 1906).

It is a very remarkable fact that in man also the oculo-motor nerve originates partly in the nuclear groups of the opposite side (Gudden, Perlia, Bernheimer, Knies, Zappert, Majano).

This is supported by pathological results (Siemerling-Boedeker) and a few clinical cases (Wishart). Wishart found in unilateral oculo-motor paralysis that the inferior oblique remained intact, whilst that of the opposite side was affected, but this indeed is an isolated case.

The root fibres of the oculo-motor nerve leave the nucleus at the ventral side, pass for the greater part through the red nucleus, and collect into a trunk between the cerebral peduncles.

The oculo-motor nucleus has relations with the optic nerve. The corresponding tracts have not yet been studied in detail.

Kölliker suggests—in agreement with Meynert and others—that the optic fibres which terminate in the anterior corpus quadrigeminum surround its cells, and that these along with their nerve processes act, either directly, or by means of numerous collaterals which penetrate into the central grey matter, upon the corresponding nuclear groups of the oculo-motor nerve. Others think that the bundles which supply the pupils have already left the optic tract. The majority agree with Gudden that there are special fibres in the optic nerve which control the pupillary light reflex, and that these become partially decussated in the chiasma, so that the optic tract contains pupillary fibres for both eyes. The views of different writers as to the further course of these fibres differ so widely, that the question, which has been specially studied by Heddaeus, Massaut, Moeli, Bechterew, Bernheimer, and Bach, will not be discussed here. Monakow thinks it probable that a system of cells for reflex transmission is interposed between the terminal ramifications of the optic nerve and the cells of origin of the pupil fibres. Very probably most of these association cells are situated in the middle grey of the anterior corpus quadrigeminum and in the part of the reticular formation which lies to the side of the central grey matter. A similar description is given by Kohnstamm, who assigns to his intratrigeminal nucleus an intercalary function (*N. C.*, 1905). See also the reports of Hippel, *M. m. W.*, 1904, and Bumke.[1]

Bach[2] is absolutely opposed to the older views. He denies the direct relations of the primary optic fibres or of the optic nerve itself to the nucleus of the oculo-motor nerve. After removal of the ciliary body and the iris he found no change in the oculo-motor trunk and nuclei. He explains this by the fact that the first neuron does not extend nearly so far, but terminates in the ciliary ganglion, in which he, Schwalbe and Massaut, Bumm, and others found cell degeneration. He agrees with Michel and Kölliker that the oculo-motor fibres going to the ciliary ganglion terminate

[1] "Die Pupillensstörungen bei Geistes- und Nervenkrankheiten," Jena, 1904, with bibliography.

[2] *Z. f. Aug.*, Bd. lix.; *N. C.*, 1903, and *M. m. W.*, 1907; also "Was wissen wir über das Pupillenreflexzentrum," Berlin, 1904 (S. Karger).

in it and spread their end ramifications round its cells. In Bach's opinion there is a pupillary reflex centre in the lowest segment of the medulla oblongata or in the superior segment of the cervical cord; long tracts must therefore lead from the primary optic nerve centre or from the optic nerve to this reflex centre. From this bulbo-spinal reflex centre comes an ascending tract to the oculo-motor nucleus or to the ciliary ganglion, probably passing through the posterior longitudinal bundle. He regards this as an inhibitory centre. Trendelnburg and Bumke, however, derived other conclusions from their experiments. The oculo-pupillary symptoms which

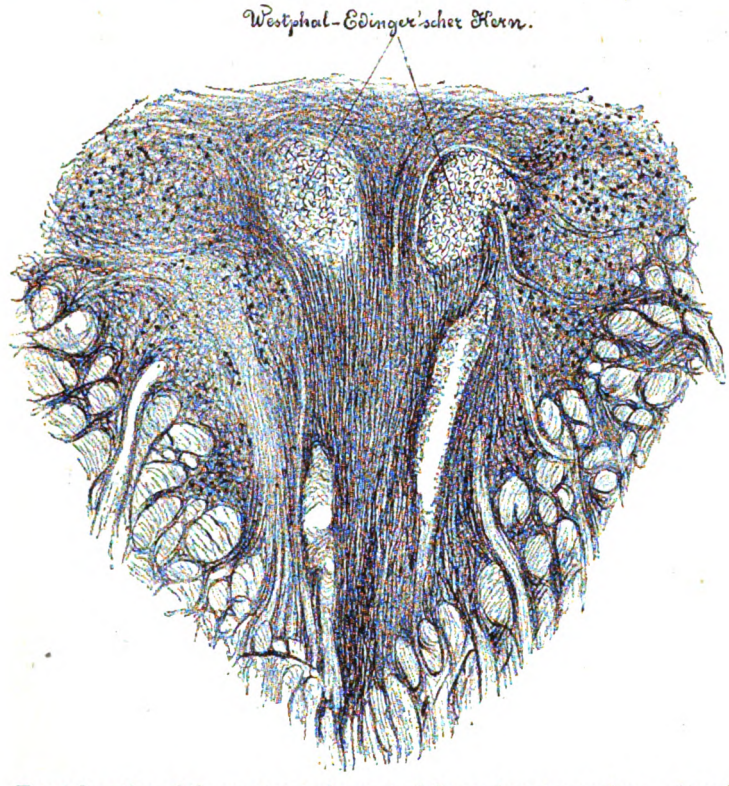

FIG. 281.—Frontal section of the most anterior part of the oculomotor nucleus. Atrophy of the Westphal-Edinger nucleus. (Carmin stain.)

occur in diseases of the medulla oblongata (Breuer-Marburg, Babinski-Nageotte) are not yet sufficiently explained.

The cause of *reflex immobility of the pupils* is probably a lesion of the centripetal pupillary fibres before their entrance into the oculo-motor nucleus (Möbius, Moeli, Uhthoff, Bumke [1]). Majano thinks that the pupil light reflex depends mainly on the crossed anterior corpus quadrigeminum. Bach (*N. C.*, 1906) is of opinion that the localisation varies, and that there is usually a lesion of the fibre systems which connect its bulbar pupillary centre with the corpora quadrigemina. This theory is opposed by Bumke and others. Schütz found the cause to be a degeneration of the dorsal longitudinal bundle which he delimits. Experimental investigations by Gudden and Bechterew show that a lesion of the floor and the lateral wall of the third ventricle may produce reflex immobility of the pupils. In one case of reflex immobility I found degeneration of the Westphal-Edinger nucleus (Fig. 281), but it was absent in another case. The cause of this symptom has been sought in various other sites, even in the ciliary ganglion (Marina). In a few cases Marburg found no change whatever in the central nervous system. We need not here discuss

[1] *Z. f. N.*, XXV.

the theory that the site of the process is localised in the cervical cord (Gaupp, Wolf, Reichardt). Bumke differs from the conclusions of these writers (*N. C.*, 1906; *Kl. M. f. Aug.*, 1907). The problem therefore still waits its solution.

Mendel[1] infers from his experiments that the oculo-facial nerve originates in the nuclear region of the oculo-motor nerve—and indeed in its distal segments. This view is supported by Spitzka, Tooth, and Turner,[2] etc., but contradicted by the investigations of Schiff-Cassirer, Siemerling, and others.

The posterior longitudinal bundle is, in the opinion of many writers, a tract which connects the various nuclei of the ocular nerves with each

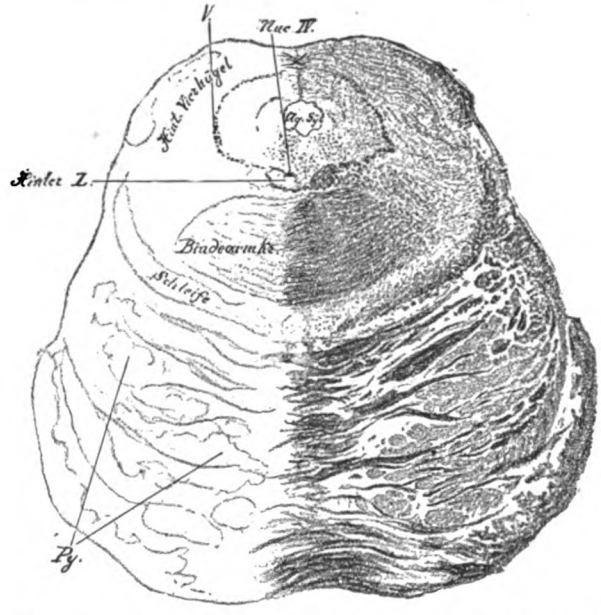

FIG. 282.—Frontal section of pons in the region of the posterior corpora quadrigemina. L. = Posterior longitudinal bundle. *Hint. Vierhügel* = Post corp. quadrig. *Nuc. IV.* = Trochlearis nucleus. *V.* = Upper cerebral root of trigeminus. *Hinter L.* = Posterior longitudinal fasciculus. *Bindearmkz* = Decussation of the superior cerebellar peduncles. *Schleife* = Fillet. *Py.* = Pyramid.

(FIGS. 282-291 are drawn from sections stained by Weigert's or Pal's methods.)

other, and also the abducens nucleus of one side with the nuclear region of the internal rectus of the other. Bechterew's anatomical investigations on the new-born child, and the cases of Bleuler, Gee and Tooth support this view, which is questioned by Panegrossi and others. Many investigators, such as Spitzer,[3] Seggels, etc., now incline to the view that this bundle connects the abducens nucleus with the oculo-motor nucleus *of the same side*, and with the groups of ganglion cells which send their root fibres to the *opposite* rectus internus. This bundle possibly receives other fibres which connect the trigeminus with the oculo-motor nerves (Mahaim, Kohnstamm), and still others which extend into the facial and hypoglossal nuclei.

[1] *N. C.*, 1887. [2] *Br.*, 1891. [3] *Obersteiner*, ix.

According to recent investigations (Ramón y Cajal, Kölliker, Held, Tschermak, Gehuchten,[1] Bruce, Kohnstamm, Turner, Fraser, Thomas,[2] Wallenberg,[3] Spiller,[4] etc.), the fibre bundle which extends from the posterior commissure or Darkschewitsch's nucleus into the spinal cord and receives tracts of ascending and descending course is of great importance, as it not only connects the oculo-motor nuclei with each other, but connects them also with Deiters' nucleus and through it with the vestibular nerve, the cerebellum, and the spinal cord. The tracts descending into the spinal cord are practically all homolateral, whilst one of the ascending tracts of this bundle from the nucleus of the anterior root zone enters into relation with the nuclei of the cranial nerves of the opposite side (Hösel). These fibre bundles therefore play an essential part in the physiological maintenance of equilibrium, orientation in space, etc. They have in particular to do with the automatic and reflex functions of controlling the movements of the eyes and the attitude of the head, of orientation in space, and the regulation of equilibrium. They also appear, previous to their extension into the nuclei of the ocular nerves, to participate in the voluntary impulses for lateral movement of the eyeballs, etc., which originate in the corresponding cortical centres. For

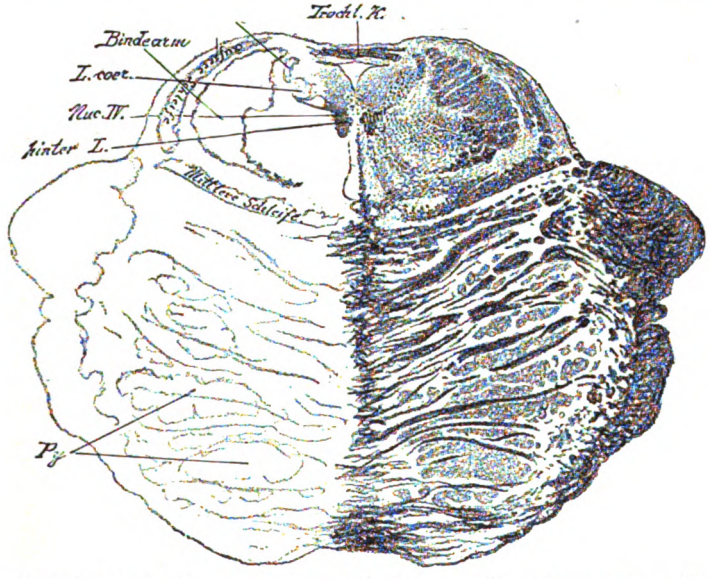

FIG. 283.—Frontal section of pons at the level of the trochlear decussation. The cerebral trigeminal root is marked V. *Trochl. K.* = Trochlearis decussation. *Bindearm* = Superior cerebellar peduncle. *aufsere Schleife* = Lateral fillet. *Mittlere Schleife* = Mesial fillet. *L. coer.* = Locus coeruleus. *N. IV.* = Trochlearis nucleus. *hinter L.* = Posterior longitudinal fascia.

special details as to the nature and cause of paralysis of conjugate deviation, see the section on symptomatology.

These facts have been established by clinical and anatomical observations, but more especially by the study of secondary degeneration, which has shown that these fibre bundles degenerate in an ascending and descending direction, and that the degenerated fibres may be traced into the nuclei of the cranial nerves, into the mid-brain, and into the spinal cord (Ramón y Cajal, Boyce, Russell, Thomas, Probst, Spitzer, Gee-Tooth, Long, Kohnstamm, Fraser, Marie-Guillain, Bruce, Münzer-Wiener, Lewandowsky).

There were formerly many differences of opinion as to the site of the *trochlear* nucleus (Westphal, Siemerling, Kausch, Pacetti, Boedeker, etc.). It is now pretty generally accepted that there is a nucleus in the posterior corpus quadrigeminum, in a bend of the posterior longitudinal

[1] *Névraxe*, 1904. [2] *R. n.*, 1903. [3] *Z. f. N.*, xxvii.
[4] *Journ. Nerv. and Ment. Dis.*, 1905.

bundle, immediately adjacent to the oculo-motor nucleus, from which this nerve takes its origin (Figs. 282-284). The root emerging from this nucleus at first assumes a dorso-lateral direction, then turns backwards and decussates in the medullary velum (Fig. 283).

According to Siemerling and Boedeker (*A. f. P.*, xxix.), and to Bach (Graefe's *A.*, 57-59, and *C. f. N.*, 1906), the decussation, as Stilling had already stated, is a partial one. Commissural fibres are also found between the two nuclei.

The area of the trigeminus nucleus is of great extent.

The sensory root of the nerve has its centre of origin, as we are now justified in assuming, in the *Gasserian ganglion*, from which it makes its way into the pons, where in the anterior or upper part of the fourth ventricle (Fig. 285) it reaches the sensory nucleus, and from there turns

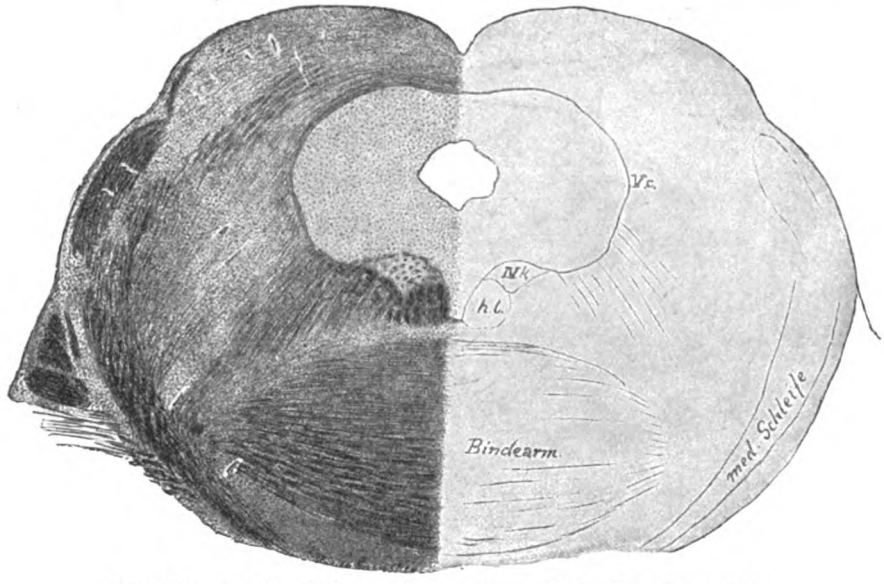

FIG. 284.—Position of the trochlear nucleus. *IV.k.* = Trochlear nucleus.

in a longitudinal or sagittal direction to pass downwards through the whole pons and the medulla oblongata as far as its lowest section, or as far as the second cervical segment (Wallenberg) in the form of a *spinal descending* trigeminal root—the bulbo-spinal tract of the trigeminal nerve. The term *spinal*, which Kölliker uses, is the least misleading.

In its course it gives off collaterals on all sides, which make their way into the neighbouring grey matter (substantia gelatinosa, which eventually turns into the posterior horn of the spinal cord) and surrounds its cells. It further sends out collaterals to the nuclei of the facial, and probably also to other motor cranial nerves, which may evoke the corresponding reflexes. Connections between the sensory root of the trigeminus and the solitary bundle are also assumed (Wallenberg, Kohnstamm). Kohnstamm speaks of a vagus portion of the trigeminus which has relations with the solitary bundle and perhaps also with the dorsal vagus nucleus. According to Wallenberg it is chiefly the fibres concerned in the conduction of the taste sense which pass in this way into the solitary bundle and reach the glosso-pharyngeal nucleus. Kölliker thinks that the so-called *sensory nucleus* of the pons is merely the upper portion of the grey matter, with which it is associated throughout its course. Kohnstamm (*N. C.*, 1905) finds an analogy between it and the

nuclei of the posterior columns, and between the roots which enter into it and the posterior columns, whilst he (and Lewandowsky) trace the origin of the so-called pontine fillet to this nucleus. He considers that this part of the trigeminal conduction has mainly to do with the sense of position (and the tactile sense), and he and Wallenberg [1] think that the spinal root and its grey matter—like the posterior horn and the antero-lateral tract of the spinal cord—serve exclusively for the conduction of pain and temperature. The secondary (bulbo-cortical) tract to which it gives rise naturally crosses at levels of the brain stem lower than those which serve for the sense of touch and the muscular sense (E. Müller).[2] The corneal reflex is also associated with this region. Kohnstamm has pointed out a portion of the trigeminus which he regards as corresponding to one of the lateral cerebellar tracts.

According to recent experiments (Bregmann, Wallenberg, etc.) the most proximal segments of the spinal root correspond to the third branch of the trigeminus, and the distal segments to the first branch. Kohnstamm thinks the ventral bundle corresponds to the area of innervation of the

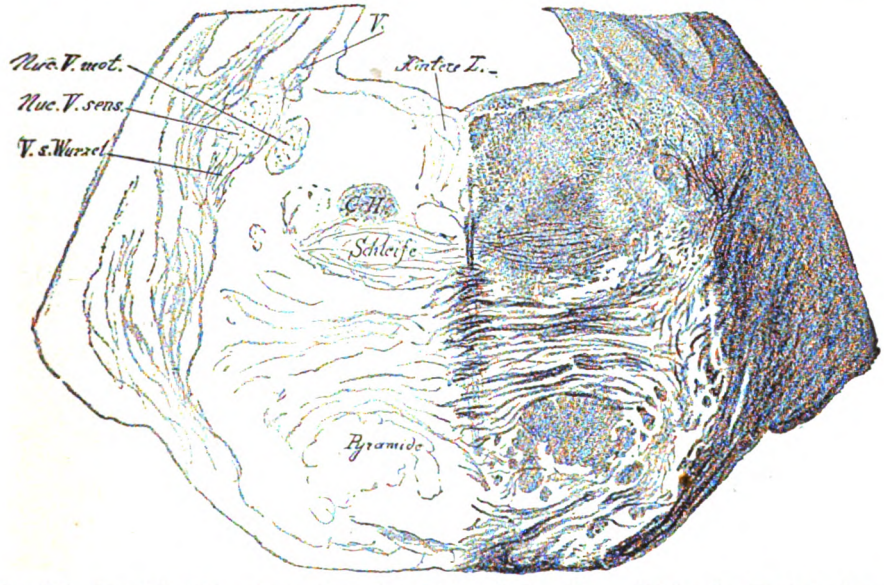

FIG. 285.—Frontal section of pons in region of trigeminal nucleus. *Hintere L.* = Posterior longitudinal fasciculus. *V.* = Upper root of trigeminus. *Nerv. V. mot.* = Motor root of trigeminus. *Nuc. V. sens.* = Sensory nucleus of trigeminus. *V. s. Wurzel* = Sensory root of trigeminus. *C. H.*—*C. H.* = Central tegmental tract. *Schleife* = Fillet.

first, or first and second branches. The branches for the mucous membrane of the mouth and tongue appear to run in the dorsal portion of the transverse section of the root, which increases in strength as it passes frontalwards. As to the secondary or central trigeminal tract, see p. 643.

The topography of sensory affections in lesions of the nucleus and roots of the trigeminus differs essentially from that in lesions of the peripheral nerves (Schlesinger, Laehr, Soelder, Müller, Kutner-Kramer).

The smaller *motor root* has its origin from the large-celled *motor nucleus* (Fig. 285).

There is further a *cerebral* (formerly called *descending*) root of this nerve, the radix mesencephalica n. trigemini—which may be traced into the anterior corpus quadrigeminum, and lies like a small crescent laterally to the aqueduct of Sylvius (Fig. 283). It has its origin in the ganglion cells which accompany the root in its course. At the level of the two nuclei, it passes between them into the emerging trigeminal root (Fig. 285).

[1] *Z. f. N.*, xxvii. [2] *Z. f. N.*, xxxi.

The function of this root has not been determined. It has been taken to be *trophic*, but in a case where I found it degenerated on both sides there were no trophic symptoms of any kind. Hagelstam's cases also contradict this theory. Kölliker, with Ferrier, Held, and others, regards it

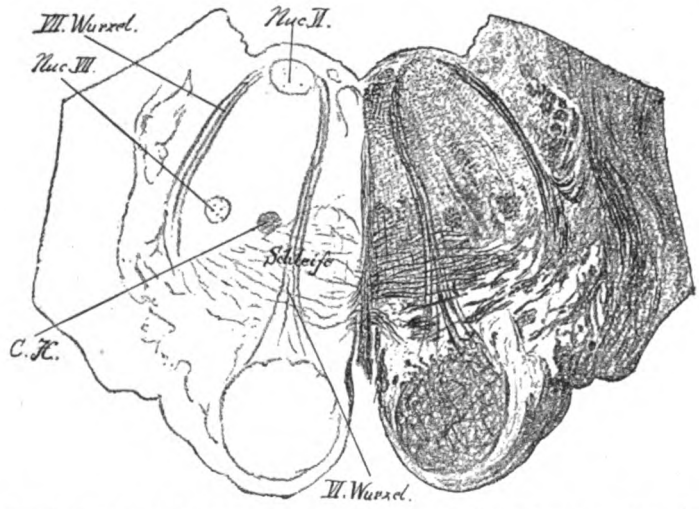

FIG. 286.—Frontal section of pons at level of abducens and facial roots. *Nuc. VI.* = Nucleus of abducens. *VI. Wurzel* = Root of abducens. *Nuc. VII.* = Nucleus of facial nerve. *VII. Wurzel* = Root of facial. *C. H.* = Central tegmental tract. *Schleife* = Fillet.

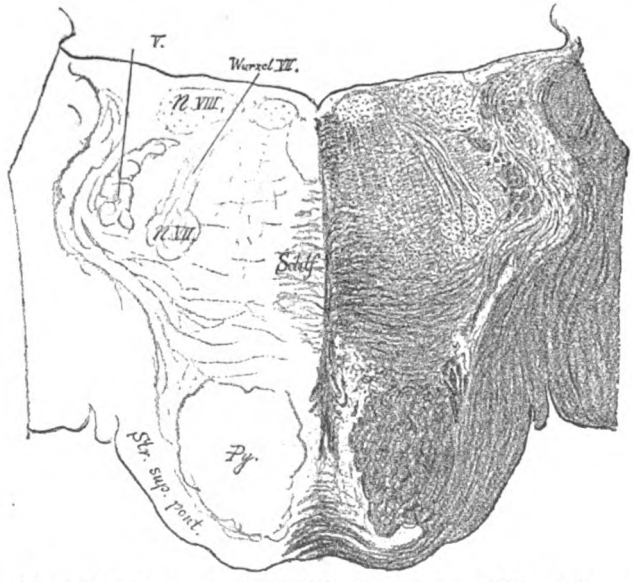

FIG. 287.—Frontal section of pons at level of facial nucleus. *Schlf.* = Fillet. *Wurzel VII.* = Root of facial nerve.

as a motor root, and suggest that it may control the innervation of the tensor tympani and veli palat. muscles. This is supported by the investigations of Teterjanz. Edinger traces its fibres into the motor root, and Probst has followed one of its bundles in the distal direction into the medulla oblongata. Ramón y Cajal thinks it sends collaterals into the motor nucleus, where they

surround its cells. Wallenberg (*D. m. W.*, 1905) thinks it is concerned in the reflex movements of feeding. Johnston (*Anat. Anz.*, 1905), who examined the root in lower animals, came to another conclusion. Kohnstamm states that the "tractus Probsti" has its origin in it, and he describes one of the nuclei it contains as the "nucleus intertrigeminalis." The statement that it belongs to the trochlearis is now regarded as definitely disproved (Gehuchten). Fibres also pass from the locus

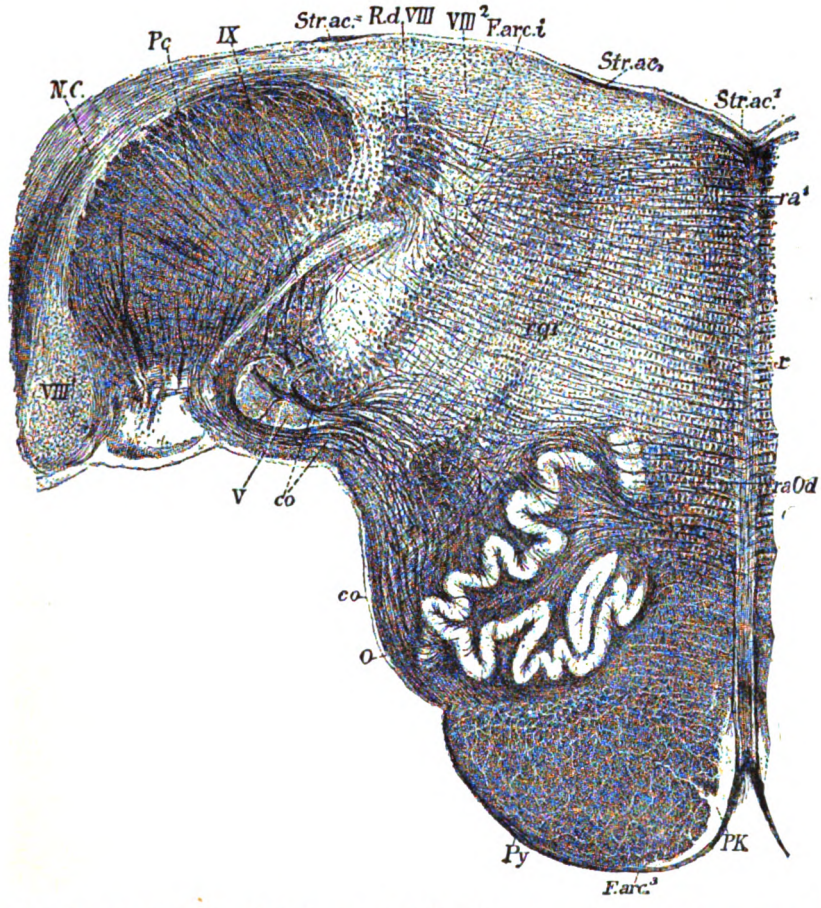

FIG. 288.—Transverse section of the human medulla oblongata at the site of the entrance of the auditory nerve. (Weigert's stain.) Enlargement 7 : 1. *VIII.*¹ = Ventral acoustic nucleus. *VIII.*² = Dorsal acoustic nucleus. *N. C.* = Cochlear nerve. *P.c.* = Cerebellar peduncle (restiform body). *R. d. VIII.* = Descending acoustic root. *Str. ac.* = Striæ acousticæ. *co.* = Cerebello-olivary fibres, etc. (After Kölliker.)

cœruleus into the motor root; they, like those originating in the motor nucleus, only enter the root of the same side (Schuzo-Kure).

286 Fig. 268 shows the position of the *abducens nucleus*. The root fibres pass uncrossed through the tegmentum and pyramids to the emerging nerves.

Gehuchten and Pacetti describe a nucleus lying ventral to the abducens nucleus, which they regard as an accessory nucleus of this nerve. Kaplan and Finkelnburg were able to confirm this.

The *facial nucleus* (Fig. 287) is situated in the inferior segments of the pons, internal to the spinal trigeminus. It is about 4 mm. in length. The roots which arise from it run first towards the floor of the fourth ventrical, in a median and cerebral direction, bend at a right angle into the so-called knee, and after a short course in the direction of the brain again turn backwards (spinalwards), make their way downwards and outwards, and from the nerve *root* which emerges between the facial nucleus and the trigeminal root. The root appears also to receive an accession of fibres from the opposite nucleus (Stieda, Obersteiner, Flatau, Wyrubow, Bary).

The relations of the various segments of this nucleus to the various muscle groups, and the nuclear localisation of the so-called superior and inferior facial, are not yet fully explained, in spite of the investigations of Marinesco, Pardo, Wyrubow, Kotelewski, Parhon, and Papinian (*Semaine méd.*, 1904). Wyrubow describes an accessory nucleus. Its relation to the facial nucleus is still doubtful.

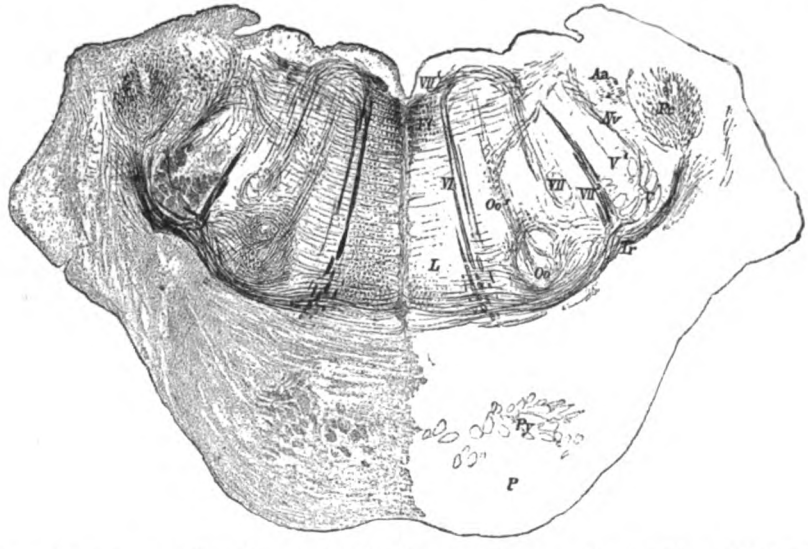

FIG. 289.—Transverse section through the distal part of the pons of an eight months' embryo. (Pal's method.) Enlargement 6 : 1. *Oo.* = Superior olive. *Oo.*¹ = Its peduncle. *L.* = Fillet. *N.v.* = Vestibular nerve. *VII.*¹ = Facial knee. *VII.*² = Emerging facial root. Pyramidal and transverse fibres of the pons still non-medullated. (After Kölliker). *R. C.* = Restiform body (inferior cerebellar peduncle). *A. a.* = Spinal acoustic root.

The *portio intermedia Wrisbergii* is supposed to originate in the geniculate ganglion. According to Duval it forms the superior portion of the sensory glosso-pharyngeal root. The chorda tympani passes by means of this nerve into the solitary bundle (Wallenberg) and the glosso-pharyngeal nucleus. Kohnstamm regards the portio intermedia as the continuation of the chorda. Nageotte has recently studied the question in detail (*R. of N.*, 1906).

Kohnstamm describes a special nucleus (or several nuclei) for the innervation of the salivary glands (nucleus salivatorius) (*Anat. Anz.*, 1902, and *A. f. P.*, xxxvii.). It lies between the facial and motor trigeminal nuclei. Its root fibres run into the so-called crossed facial root or into the portio intermedia.

The *acusticus* consists of two roots : the *cochlear nerve*, which is the auditory nerve proper, and the *vestibular nerve*. The former, which rises in the spiral ganglion of the cochlea, forms the outer (posterior) root

(Fig. 288, *N. C.*); it lies at the outer side of the restiform body and terminates almost entirely in the *accessory* or ventral *acoustic nucleus* (the VIII.[1] acustic central ganglion of Kölliker) and in the tuberculum acusticium. These nuclei are connected with the *trapezoid body* and the superior olive (Fig. 289), mainly of the opposite side, from which the *lateral* fillet takes its rise. Auditory impressions are now generally supposed to pass through these paths and then through the lateral fillet, the posterior superior peduncle, the median geniculate ganglion and optic thalamus, to the cortex of the temporal lobe.

Secondary degeneration has sometimes been found in the lateral bundle of the foot of the cerebral peduncle and in the lateral fillet in old foci in the temporal convolution.

The *vestibular nerve*—the anterior, median root, which originates in the labyrinth (ganglion Scarpæ)—has probably no auditory functions, but conducts impressions influencing co-ordination to the central organs,

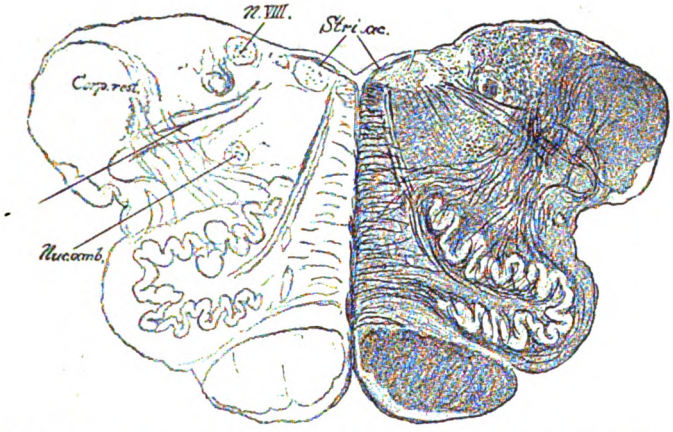

FIG. 290.—Transverse section through medulla oblongata at the level of the commencing acoustic nucleus. (Weigert's method.) *Stri. ac.* = Striæ acusticæ. *N. VIII.* = Dorsal acoustic nucleus. *Nuc. amb.* = Nucleus ambiguus. *Corp. rest.* = Restiform body.

especially to the cerebellum and to the nuclei of the ocular nerves, etc. It terminates probably partly in the *dorsal, medial auditory nucleus* (main nucleus) (Fig. 288), partly in *Deiters' nucleus* and in its lateral and cerebellar continuation, and the *vestibular nucleus* (Bechterew).

The term "Bechterew's nucleus" should be entirely abandoned as the name is applied by different writers to absolutely different structures (Kohnstamm). The vestibular roots enter into an extensive nuclear area in the latero-ventral margin of the fourth ventricle, the *angular* nucleus, and different portions of the grey matter of this nucleus may be defined as the nucleus of Deiters, supremus and medialis (Kohnstamm, personal communication). Lewandowsky has named the ventro-caudal segment the "griseum fasciculorum Rolleri."

These nuclei are connected with the cerebellum, spinal cord, and nuclei of the ocular nerves. The name of nucleo-cerebellar tracts is given to those which pass uncrossed from the nucleus tecti to the nucleus of Deiters, and also to those doubly crossed and therefore homolateral tracts between the dentate nucleus—red nucleus—and Deiters' nucleus, and thence to the spinal cord. The tracts which go to the abducens and oculomotor nuclei take their course through the posterior longitudinal bundle. The vestibulo-spinal tracts to the spinal cord also pass for the most part through the posterior longitudinal bundle. Kohnstamm describes a path from the cerebellum to the nuclei of the ocular nerves through the superior peduncle, and the crossed nucleus pontinus

tegmenti (reticul. tegmenti of Bechterew), and thence to the ocular nuclei of the opposite side, thus connecting the dentate nucleus with the ocular nuclei of the same side (?).

On this subject consult also p. 645, and the works of Bonnier (*Presse méd.*, 1903; *R. n.*, 1904),

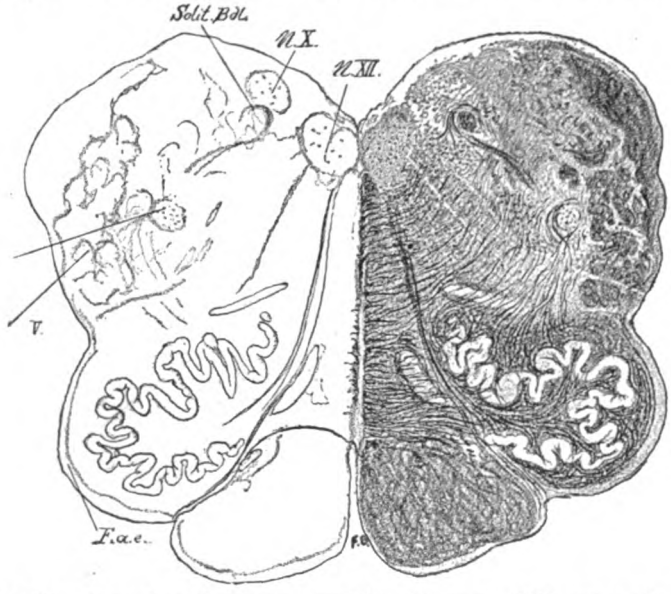

FIG. 291.—Frontal section through medulla oblongata. (Weigert's method.)

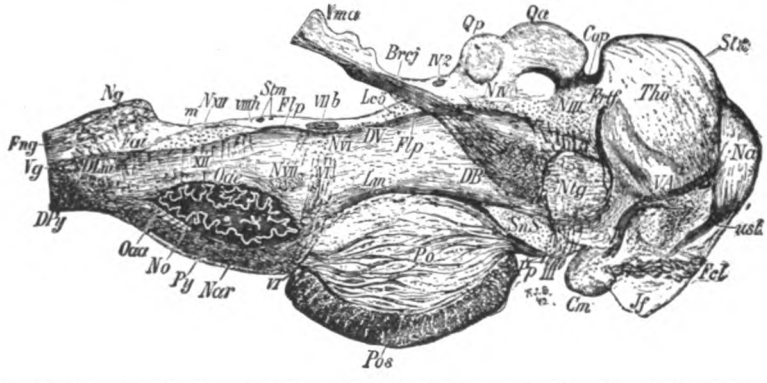

FIG. 292.—Combined sagittal section through the brain-stem. Pal's stain. (After Obersteiner.) *III.* = Oculo-motor nerve. *IV.*² = Decussation of the trochlear nerve. *VI.* = Abducens nerve. *VII. b.* = Ascending facial limb. *XII.* = Hypoglossal nerve. *Brcj.* = Brachium conjunctivum (sup. cerebell. ped.). *D. B.* = Decussation of the brachium conjunctivum. *Fai.* = Internal arcuate fibres. *Fcl.* = Pillars of the fornix. *Flp.* = Posterior longitudinal fasciculus. *Lm.* = Fillet. *No.* = Olive. *N. III.* = Oculomotor nucleus. *N. IV.* = Trochlear nucleus. *N. VI.* = Abducens nucleus, etc. *Po.* = Pons. *Pp.* = Pes pedunculi. *Py.* = Pyramids. *Fng.* = Funiculus gracilis. *N. g.* = Nucleus gracilis. *Tho.* = Optic thalamus. *Qa.*, *Qp.* = Anterior and posterior corpora quadrigemina. *Ntg.* = Red nucleus. *Cop.* = Posterior commissure.

Thomas-Egger (*R. n.*, 1903), Gehuchten (*Névraxe*, 1903), Steyskal (*W. kl. R.*, 1904), Winkler, "The Cerebral Course of the N. Octavus," etc., Amsterdam, 1907, Bárány, Clarke and Horsley, etc.

This is not the place to discuss the differences in the results of investigation on the auditory nerve made by Held, Tschermak, Thomas, Wyrubow, and others in different animals, nor to

describe the histological details of the cell and fibre connections in the auditory nucleus studied by Held, Gehuchten, and especially by Ramón y Cajal.

External to the dorsal auditory nucleus there lies a loosely compacted fibre bundle, which passes towards the spinal cord. This forms a direct (?) continuation of the funic. cuneat. of the

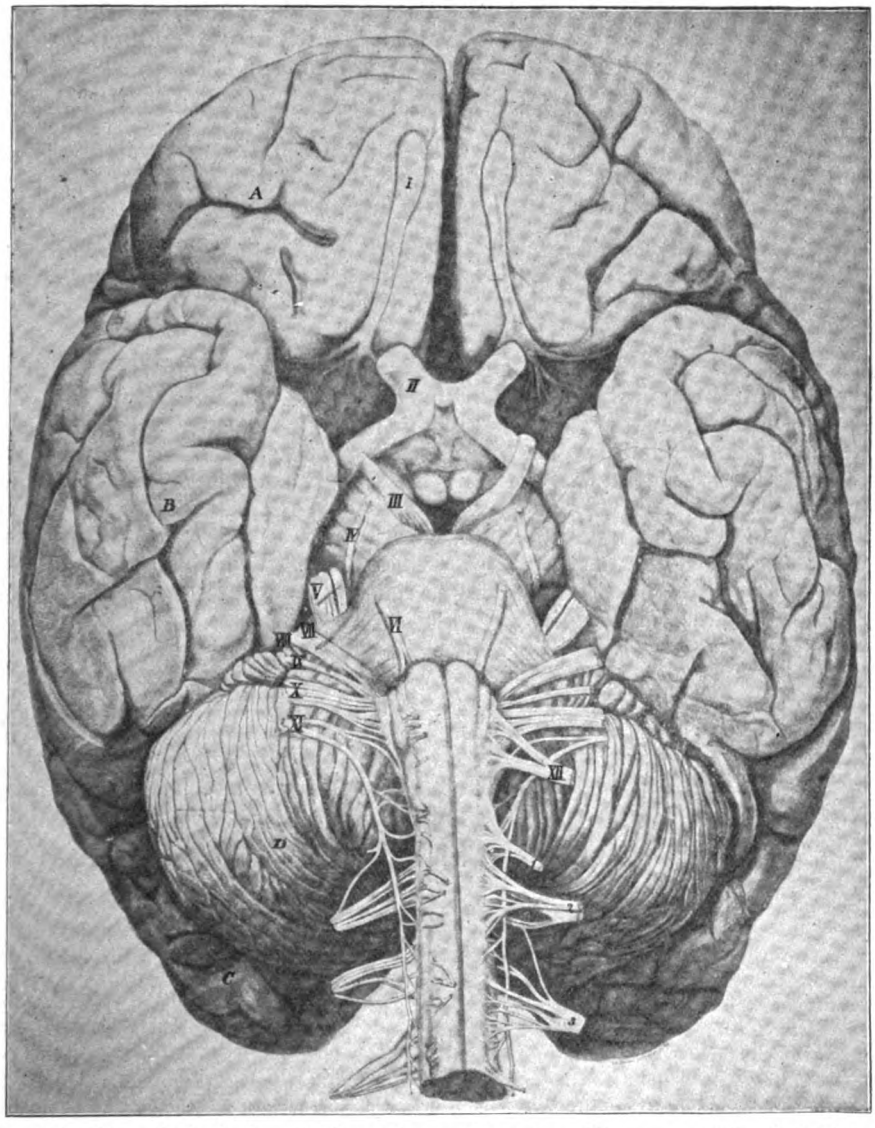

FIG. 293.—Base of the brain. Origin of the cranial nerves. These are numbered in Roman figures. *A.* = Frontal lobe. *B.* = Temporal lobe. *C.* = Occipital lobe. *D.* = Cerebellum.

spinal cord, and perhaps represents a descending auditory root (Roller). The vestibular nerve seems to be partly continued in it.

The striæ acusticæ originate chiefly from the tuberculum acusticum. They apparently represent a sensory tract of the second order, which perhaps goes as far as the lateral fillet after decussation in the raphe. The relation of these fibres to the function of hearing is entirely denied by some writers.

The *vagus and glossopharyngeus* cannot be sharply separated from each other, especially in their intramedullary roots.

The *sensory vago-glossopharyngeal root* arises from ganglia which lie outside the medulla (gangl. jugulare, petros. nodos.). It penetrates into the medulla oblongata and there forms the *solitary bundle*, the vagus root formerly known as ascending, which is in reality a descending, sensory vago-glossopharyngeal root. Its position is shown by Figs. 290 and 291.

Fibres are also said to pass into the solitary bundle from the geniculate Gasserian ganglia.

The fibres which it contains give off collaterals at every level, and these penetrate into the accompanying grey matter. The column therefore forms a terminal nucleus of the glosso-pharyngeal and vagus.

The nucleus funicul. solitar. is regarded by Kohnstamm and Wolfstein (*Journ. f. P.*, viii.) as the sensory vagus nucleus or the sensory vago-glossopharyngeal nucleus. The nucleo-cortical tracts which arise from it, join the secondary trigeminal tract. Lateral from this nucleus, a parasolitary nucleus may be demarcated. See also Hudovernig, *Journ. f. Psych.*, x.

Another portion of the sensory root of the vagus and glosso-pharyngeus penetrates into the so-called *posterior vagus nucleus* on the floor of the fourth ventricle, which is regarded as a sensory end nucleus (His, Kölliker, Held). Some writers, such as Marinesco, Bunzl-Federn, Bruce, Gehuchten, Kohnstamm, have, however, ascribed motor functions (for non-striped muscles) to this nucleus. Kohnstamm names it the nucleus sympathicus medullæ oblongatæ (see p. 497) and traces its root fibres into the vagus of the same side, not into the glossopharyngeus. He also includes in the sensory vagus region the dorsal grey matter, the "griseum dorsale areæ vagi."

The *motor nucleus of the vagus-glossopharyngeus* is probably the nucleus ambiguus (Fig. 290), from which emerge the branches which supply the muscles of the larynx and throat. This view is held by Grabower, Dees and Grossmann, Ossipow, Gehuchten, Ranschoff, Kohnstamm, Wallenberg, L. R. Müller,[1] Kohnstamm-Wolfstein,[2] and others, but it is opposed by Holm, Oordt, Monakow, and others (see p. 497).

My experience in cases of amyotrophic lateral sclerosis is in favour of this interpretation of the nucleus ambiguus.

Wallenberg localises the centre of deglutition in the proximal, and the centre for the larynx in the distal segments of this nucleus. Breuer and Marburg are of the same opinion. Fibre bundles also pass from the nucleus ambiguus into the opposite vagus root. This is specially the case as regards the fibres for innervation of the palate. I have seen in one case an acute onset of paralysis of the right palate with loss of reflexes and paralysis of the left vocal cord, a combination which is difficult to explain, even by this view. Kohnstamm and Wolfstein found that the nucleus ambiguus was always degenerated after bilateral section of the recurrent.

A small nucleus of large cells is found in the proximal part of the solitary bundle, where this tract terminates; it may perhaps represent a special motor nucleus for the glossopharyngeal. As to the relations with the intermediary nerve of Wrisberg and the taste function, see p. 477, the paper by Nageotte in the *R. of N.*, 1906, and that of Kohnstamm-Wolfstein.

Other fibres make their way from the end nucleus and the cells of the solitary bundle to the opposite lemniscus. The vago-glossopharyngeal nucleus is, according to Edinger, connected with the cerebellum, and according to Lewandowsky and Muskens, with the vestibular nerve.

The vagus depressor nerve has its origin in the jugular ganglion and extends into the dorsal vagus nucleus (Kohnstamm-Wolfstein). These writers, with Gad and Marinesco, regard the

[1] *A. f. kl. M.*, Bd. lxxxvi. [2] *Journ. f. Psych.*, viii.

lateral nucleus of the medulla oblongata, and also the nucleus funicul. solitar. and the nuclei of the reticular formation as a respiratory centre. The bulbo-spinal respiratory tracts have their course in the antero-lateral column of the same side (Kohnstamm-Rothmann). This bundle of fibres is identical with the fasciculus of Thomas.

With regard to the position of the nucleus of the spinal-accessory nerve see p. 128.

Immediately beside the raphe lies the *hypoglossal nucleus*, richly provided with fine, large cells and a thick fibre network (Figs. 291 and 276). Its roots pass between the olive and pyramids, and leave the medulla at this point. A group of cells (Roller) lying ventral to the twelfth nucleus seems to have nothing to do with the hypoglossal nerve.

Parhon and Goldstein (*Roum. méd.*, 1899) and Parhon-Papinian (*Sem. méd.*, 1904) think they have been able to define distinct groups of ganglia in the hypoglossal nucleus for the different muscles, and particularly a small external group for the ramus descendens hypoglossi.

Fig. 292 shows the position of the nerve nuclei and the course of the nerve roots.

As to the emergence of the cranial nerves from the base, consult Fig. 293.

In regard to the development of the cranial nerves, A. Westphal (*A. f. P.*, xxix.) has demonstrated the following interesting fact: At birth the medullated motor, the sensory, and mixed nerves, with the exception of the auditory, are still non-medullated. The optic nerve is the least advanced. In the third week the formation of medullary sheaths has already made obvious progress.

The Vascular Supply of the Brain

All the arterial vessels of the brain are derived from the *carotid* and *vertebral* arteries.

The *basilar artery*, which takes its rise from the junction of the *vertebral arteries*, gives origin to the *posterior cerebral arteries*. As the *posterior communicating artery* establishes a connection with the carotid, and the *anterior cerebral arteries* which arise from it are connected by the *anterior communicating artery*, the *circle of Willis* is thus formed, which encloses the chiasma, the tuber cinereum, and the corpora mamillaria (Fig. 294).

From the circle of Willis and the roots of the three large cerebral arteries (anterior, middle, posterior), there arise, during their basal course, branches which penetrate into the brain substance and, without anastomosing, give off branches to the central ganglia and the surrounding white substance.

On the rest of the surface of the brain, the arteries ramify in the pia, and from this pial vascular zone, in which the neighbouring vessels are connected by anastomoses, short and long branches make their way into the cortex, and the latter pass into the subcortical white matter. Thus we have a *central* and a *cortical* arterial plexus, which, apart from capillary network, are not interconnected by anastomoses.

From the *arteries of the Sylvian fissure* (middle cerebral artery) arise, shortly after their origin, the arteries for the basal ganglia and the internal capsule, with the exception of its posterior segment. Some of these make their way through the lenticular nucleus into the internal capsule, others pass outside the lenticular nucleus. One of the latter is the most important source of cerebral hæmorrhage. Those which go to the corpus striatum are known as the *lenticulo-striate arteries*, those which supply the optic thalmaus as the *lenticulo-optic arteries*. There are absolutely no anastomoses here. From the carotid or median cerebral artery comes a fine branch—the *choroid artery*—which runs along the optic tract and reaches the choroid plexus of the inferior cornu of the lateral ventricle. It sends off branches to the posterior part of the internal capsule.

The *posterior cerebral artery* supplies the occipital lobe (for the most part), the posterior part of the optic thalamus, and sends out branches to the tegmentum, the cerebral peduncle, the corpora quadrigemina, and to the nucleus of the oculo-motor nerve. The most anterior groups of the

oculo-motor nucleus are supplied by a special branch. The internal arteries of the peduncle and oculo-motor nuclei are terminal arteries.

The posterior cerebral artery therefore innervates both the visual and the oculo-motor centres. It sends to the cortex of the occipital lobe the occipital artery, from which arise the arteries of the parieto-occipital, calcarine, and cuneate fissures.

The *anterior cerebral artery* supplies the frontal lobe, with the exception of the third frontal

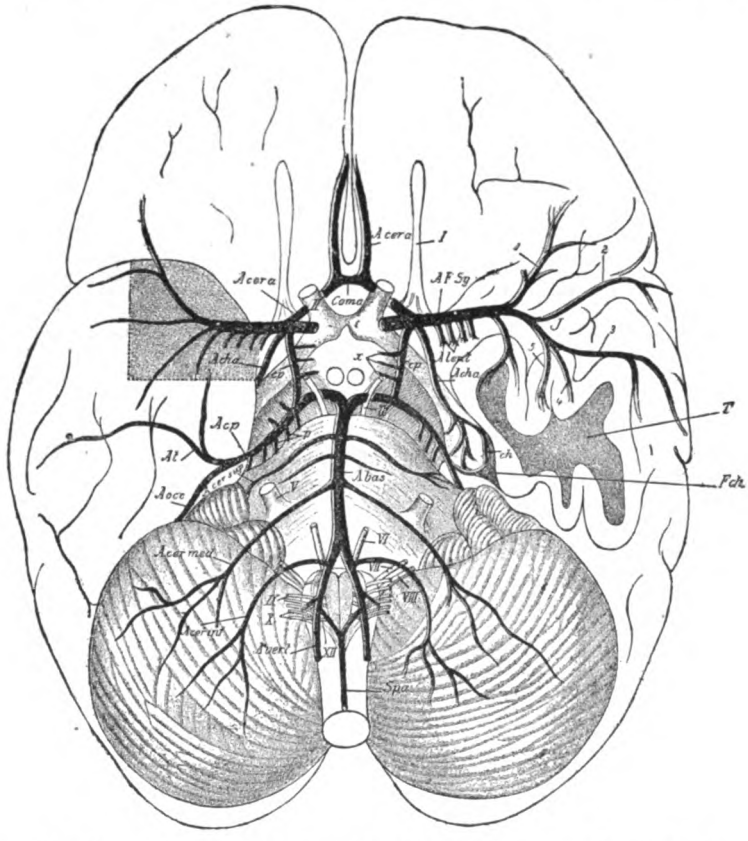

FIG. 294.—(After Monakow). The arteries at the base of the brain. *T.* = Separated temporal lobes. *A. cer. a.* = Anterior cerebral artery. *Com. a.* = Anterior communicating artery. *AFSy* = Artery of the Sylvian fissure. *A. lent.* = Lenticular artery. 1-5 = Cortical main branches of the Sylvian artery. *cp.* = Posterior communicating artery. *Acha.* = Anterior choroid artery. *x.* = Lateral branches of the posterior communicating artery. *Acp.* = Posterior cerebral artery. *p.* = Short branches of post-cerebral artery. *A. bas.* = Basilar artery. *At.* = Temporal artery (Duret). *A. occ.* = Occipital artery (Duret). *A. cer. sup.* = Superior cerebellar artery. *A. cer. med.* = Median cerebellar artery. *A. cer. inf.* = Inferior cerebellar artery. *Spa.* = Anterior spinal artery.

and anterior central convolutions, and the cortex of the median wall of the hemisphere as far as the præcuneus, as well as the corpus callosum.

The *middle cerebral artery* divides above the island of Reil into four or five branches (Fig. 295), the first of which supplies the third frontal convolution, the second chiefly the facial and arm centre on the anterior central convolution, the third the posterior central convolution and the parietal lobe, whilst the fourth conveys the blood to the area of the inferior parietal lobe, and the fifth to the most superior temporal convolutions. The cortical branches of the artery of the Sylvian fissure appear to anastomose with each other with a degree of freedom which varies in different cases.

The pons, medulla oblongata, and cerebellum derive their blood supply from the vertebral and basilar arteries. The inferior cerebellar and anterior spinal arteries, which have their origin from the vertebral artery, give off branches for the medulla oblongata. The basilar artery gives origin to an inferior and a superior cerebellar artery. The branches of the basilar and vertebral are terminal arteries.

The arterioles which supply the pons and oblongata divide into the arteriæ medianæ seu nucleorum and the arteriæ radiculares. The former pass in the middle line to the nerve nuclei, the latter run with the nerve roots and give off one branch which accompanies these to the periphery, and another which makes its way with them into the nucleus. The nerve roots arising from

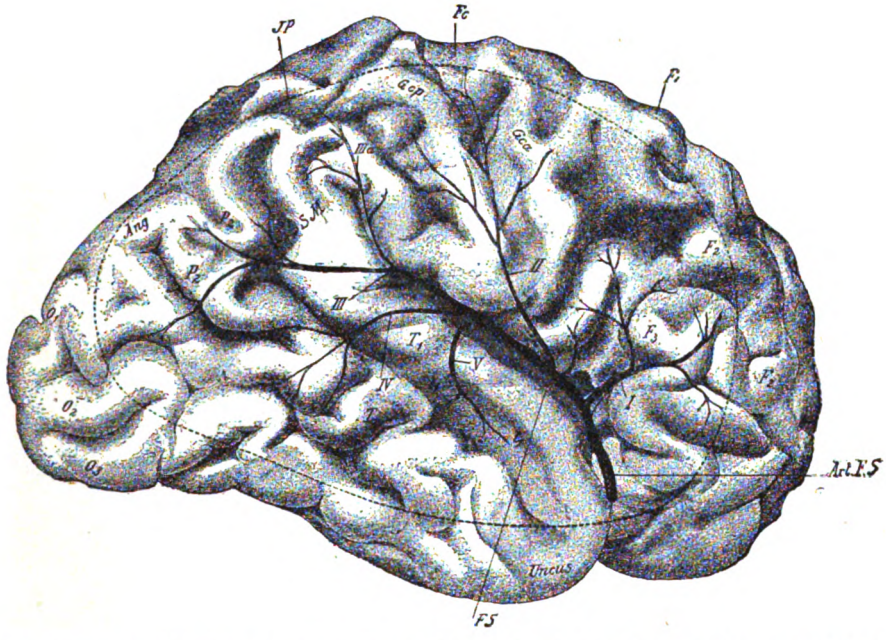

FIG. 295.—(After Monakow.) Lateral view of the right hemisphere, showing the area supplied by the artery of the Sylvian fissure. *Art. F. S.* = Artery of the Sylvian fissure. *I.-V.* = The five main branches of this artery. *F. c.* = Central fissure. *F. S.* = Sylvian fossa. *O. P.* = Interparietal fissure. *T.* = First temporal fissure. F_1.-F_3. = First, second, and third frontal convolutions. *S. M.* = Supramarginal gyrus. *Ang.* = Angular gyrus. O_1.-O_3. = First, second, and third occipital convolutions.. The dotted line shows the area supplied by the Sylvian artery.

the medulla oblongata are, with the exception of the hypoglossal, whose roots are innervated by the anterior spinal artery, mainly supplied by the vertebral.

The branches for the facial and auditory nerves may take their rise from the vertebral or basilar arteries, or from both of these. From the anterior spinal artery branches pass to the raphe, to the interolivary layer, and to the hypoglossal.

The left vertebral is usually larger than the right.

The branches for the olive and pyramid are derived from the vertebral or the anterior spinal; those for the restiform body from the inferior cerebellar arteries, which also give off branches for the roots of the vagus, accessorius, the spinal trigeminal root, and the formatio reticularis.

Heubner and Duret have given us most careful investigations on this subject, and Kolisch, Léon d'Astros, Shimamura, Rossolimo, Wallenberg, and others, have recently contributed to its study.

Wirubow (*Obosren. psich.*, 1901, abs. in *N. C.*, 1903) gives interesting details as to the anomalies of the circle of Willis.

Secondary Degeneration in the Brain

On the whole, the rules already given as applying to the spinal cord hold good here also. In the first place it is certain that nerve processes separated from their cells of origin rapidly degenerate. Destruction of the motor region therefore produces degeneration of all the motor nerve tracts to which it gives rise in the internal capsule, the cerebral peduncle, the pyramids of the pons, the medulla oblongata, and in the anterior pyramidal tract of the same side and the lateral pyramidal tract of the opposite side of the spinal cord. Thus after any lesion which interrupts the motor nerve tract at any site, the portion of those fibres distal to the focus of affection will degenerate. Isolated fibres in the lateral pyramidal tract of the same side may also degenerate.

With regard to the subcortical segments of the brain (or grey masses) and their dependence on the cerebrum as revealed in secondary degenerations in cerebral lesions, Monakow [1] makes the following divisions: 1. the *direct cerebral portion*, for whose existence the integrity of the cerebrum is absolutely necessary, and which totally degenerates and disappears when the cerebrum is destroyed; this includes the nuclei of the optic thalamus, the geniculate body, etc.; 2. the *indirect cerebral portion*, *i.e.* the segments over which the cerebrum exerts a powerful nutritive influence, but which possess a certain individuality on account of other relations. These show merely a moderate general diminution after removal of the cerebrum. This portion includes the red nucleus, the roof of the corpora quadrigemina, etc.; 3. *autochtonous subcortical centres*, which do not degenerate even after the destruction of a whole hemisphere, such as the nuclei of the motor cranial nerves, etc.

The secondary changes in the nerve cells, which are separated from their nerve processes, described on p. 124, are also observed in the brain. This is specially the case in young persons, or in affections which date from early childhood. Thus Monakow states that after section of the peduncles in young animals the giant pyramidal cells belonging to it are destroyed. Marinesco has observed this also under other circumstances.

The more exact conditions under which cellulipetal degeneration occurs are not yet explained. If the axis cylinder is divided at a great distance from the cell, the continued existence of the cell, according to Forel, is less affected than if the division takes place immediately beside it, because in the latter case the collaterals, upon which the cells can still put forth a certain amount of activity, are likewise affected.

Monakow gives the name of *secondary atrophy of the second order* to changes which consist not in severe disintegration, but in simple diminution in size of all the parts (probably due to the greatly diminished activity). To this class belongs, for instance, atrophy of the fillet after extensive and early acquired defects in the central and parietal lobes of the cerebrum, atrophy of the optic nerve after old foci in the occipital lobe, and atrophy of the external geniculate body after enucleation of the eyeball. After having persisted for years, however, this atrophy may undergo regressive changes and completely disappear (Henschen). The cortical fillet only atrophies after foci in the region of the central convolutions, when the focus is so extensive that almost the whole white matter of the motor zone and the parietal lobe of the hemisphere is also affected, and when it is acquired early (Monakow). Old foci which destroy one side of the tegmentum, produce ascending and descending degeneration of the fillet; the latter is known as retrograde. According to the unanimous statements of Dejerine, Monakow, Schlesinger, and Probst, ascending degeneration does not extend beyond the optic thalamus.

We need not discuss here the question of ascending and descending degeneration in the posterior longitudinal bundle, in the superior cerebellar peduncle, the formatio reticularis, in the rubro-spinal, vestibulo-spinal, and other systems of fibres.

[1] "Gehirnpathologie," 2nd ed., Wien, 1905. Consult the bibliography here.

GENERAL SYMPTOMATOLOGY OF BRAIN DISEASES

I. General Symptoms

We shall here consider only the organic diseases of the brain; functional diseases will be discussed in a special section.

The morbid symptoms are determined less by the nature of the process than by the site of the disease.

Certain symptoms may appear in diseases of every area of the brain. We shall distinguish these as *general brain symptoms* from the *focal symptoms, i.e.* from those signs of brain disease which are produced by lesion of a certain part.

The diagnosis of brain disease is founded upon the general symptoms. They also give most information as to the nature of the anatomical process. The focal symptoms point to the *site* of the lesion; they enable us to localise the brain disease.

Among the general symptoms of brain disease we include: *headache, vomiting, vertigo, affections of consciousness and the mind, changes of the pulse and respiration, rise of temperature, etc. Convulsions*, which may indeed belong to the general symptoms, but are more often to be regarded as a focal symptom, will be discussed later.

Choked disc, the most important sign of increased brain pressure, will be dealt with under affections of the optic nerve.

Headache is one of the most common symptoms of brain disease. Its diagnostic importance is, it is true, considerably diminished by the fact that it may be evidence not only of organic, but also of functional disease of the brain, and even of the most varied diseases of other organs, and that it is impossible to assign any definite indications by which a headache due to organic brain disease can be sharply distinguished from one due to other causes. It is only the association of this symptom with other cerebral signs that points to the site and nature of the lesion. One may say very generally that a particularly intense and persistent headache is usually caused by an organic brain disease, but even this statement is by no means absolutely valid, as hysterical and neurasthenic people often complain of persistent violent headache, and severe, intractable headache is apt to develop from migraine, or as a modification of it, and sometimes from certain intoxications.

The headache due to brain disease is usually aggravated by coughing, straining, or sneezing, but so is the headache due to affections of the circulation, especially venous congestion. Many forms of neurasthenic headache show this characteristic.

The headache of hysteria, and to a certain extent of neurasthenia, is usually characterised by being directly dependent upon mental influences and morbidly exaggerated self-observation. As to other diagnostic symptoms, see the corresponding chapter.

The headache which accompanies organic brain disease is either diffuse, felt over the whole head or many parts of it, or it is localised. In the latter case the site of the disease usually fairly corresponds to the region of the headache, but it may be felt in the frontal region, in diseases of the occipital lobe, and *vice versâ* in the nape of the neck in affections of the frontal lobe.

The headache caused by chronic poisoning may in every respect correspond to that due to organic disease of the brain.

A combination of headache and *vomiting* points to an organic lesion, if fever, intoxication, uræmia, migraine, and a morbid gastric condition can be excluded. Vomiting comes on, as a rule, at the height of the headache. It is usually characterised by the ease with which the vomiting takes place. As a rule there is neither abdominal pain nor retching movements, and often not even a long stage of discomfort; the contents, of the stomach are simply evacuated suddenly and with ease. This is, however, not always the case. The vomiting does not depend upon the food taken; it may occur when the patient is fasting. It may also follow a meal, and be specially provoked by eating. Thus I have seen cases in which this irritability of the vomiting centre made it impossible to give any food during the attacks, which lasted hours or days. It may be brought on by a change of position, such as raising the head. Although this symptom may occur in disease of every region of the brain, it is particularly marked in affections of the cerebellum and medulla oblongata.

Cerebral vomiting is not as a rule associated with intestinal troubles, but it may, in my experience, be combined with diarrhœa. The combination with nasal hæmorrhage, which I have occasionally seen, was probably due to a special predisposition to the latter and to the increase of pressure produced in the vessels of the nose.

Vertigo is a symptom of little diagnostic importance. Although this term is applied to symptoms of very different kinds, it conveys as a rule the idea of a *sensation of the loss of balance*, coming on suddenly and again rapidly vanishing, or persisting for some time. The patient feels as if the ground were swaying beneath his feet, as if everything were turning round him, or as if his own body were in a state of rotation. A very transient affection of consciousness is often referred to by the patient as vertigo. This symptom may have so many causes (see chapter on vertigo) that it is usually only of diagnostic importance when associated with others. A specially severe and obstinate form of vertigo occurs in diseases of the cerebellum, the cerebellar peduncle, and the vestibular nerve, and tends here also to produce objective signs of disordered equilibrium. It is brought on or exaggerated by rising from the horizontal position or by changing the position of the head. A similar kind of vertigo is caused by affections of the labyrinth. We shall return to this symptom and the methods of testing it.

Affections of Consciousness, Mental Symptoms.—Even if we except the psychoses in the strict sense of the word, mental disorders are very common in organic brain diseases. They are less often produced by a circumscribed disease, which acts upon the brain either directly or by means of pressure, than by diffuse morbid processes which extend their influence over the whole of the brain. Diseases of the frontal lobes seem specially calculated to give rise to mental troubles (see p. 631).

In brain disease the mind may be affected in many ways. According to the severity of the affection we distinguish between simple *stupor*, *somnolence* (the patient is drowsy, but can easily be wakened, only to fall back into light slumber or an apathy which resembles sleep), *sopor* (the patient can only be roused by strong sensory stimuli), and *coma* (complete loss of consciousness, usually with absence of reflexes and tendon phenomena, from which the patient cannot be roused). There

are, however, various stages of coma, in which the condition of the different reflexes varies. It is only in the deepest stage that they are all abolished. In apoplectic (and epileptic) coma it has been found by Babinski, Brissaud (*R. n.*, 1902), Oppenheim, and others, that when the other tendon reflexes are lost the plantar reflex may correspond to Babinski's type, and it is under these conditions also that the dorsal leg-phenomenon has been found by Oppenheim, Pfeifer (*M. f. P.*, xiv.), and others.

There is sometimes in brain disease a condition (*trance*) which can scarcely be distinguished from sleep, except that it may last for days, weeks, and months.

It is exceedingly difficult to recognise slight degrees of stupor which may easily be taken for mental weakness. We may notice that the patient thinks clearly and shows correct judgment whenever he gives his attention to the matter in hand, but that he has difficulty in collecting his thoughts, has to make an effort to keep his attention fixed, is given to staring into vacancy, and takes little part in what is going on round him. It is a very significant sign of mental disturbance when the patient passes urine and fæces involuntarily, although the sphincter functions are normal. In severe forms of stupor the patient may forget to masticate and swallow, and may retain the food for a long time in his mouth, whilst in coma he does not swallow at all.

In a few cases of coma in which the reflexes were not completely lost, I have seen a peculiar symptom, viz., exaggeration of the reflex movements of sucking, masticating, and swallowing. Stroking the lips was sufficient to produce a number of rhythmic movements of this kind. For details of this symptom, which I have named the " feed reflex " (*M. f. P.*, xiv., and *B. k. W.*, 1904), see the chapter on spastic diplegia, pseudo-bulbar paralysis, etc.

Stupor is the name given to a condition of marked impairment of consciousness in the waking state in which the patient has entirely or almost entirely lost consciousness of his relation to the outer world. The reflexes are preserved.

Chronic brain disease, which extends over a large area or affects the whole brain by means of pressure (*e.g.* tumour), may cause gradually increasing mental disorder, which in the end becomes loss of consciousness. Sudden loss of consciousness may be due to cerebral hæmorrhage or vascular obstruction, but injury (concussion of the brain) or violent mental excitement may also produce loss of consciousness. The unconsciousness of the epileptic attack is of sudden onset ; that due to loss of blood (anæmia of the brain) and intoxication as a rule develops gradually.

The sudden onset of complete loss of consciousness is known as *apoplexy* from the sudden falling down of the patient, but it has become more and more customary to give this name to the coma caused by cerebral hæmorrhage. As the same symptoms may result from embolism of a cerebral artery, the idea of apoplexy does not exclude this cause.

Cerebral hæmorrhage gives rise in rare cases to an impairment of consciousness which gradually develops into coma (apoplexia ingravescens or progressiva). The special characteristics of an apoplectic attack will be described in the chapter on cerebral hæmorrhage.

I have described under the name of " Lachschlag " (laughing attack) (*M. f. P.*, xi.) an uncommon symptom observed by myself and once by Binswanger, viz., that in laughing,

a condition of unconsciousness comes on, at the height and as the result of the laughter, which usually lasts only for a short time.

Delirium is a most characteristic form of mental disturbance. This is a condition of excitability and confusion, due to sensory delusions and transitory hallucinations, associated with motor restlessness. But delirium is much less often the result of organic brain diseases than of *fever*, *intoxication* (alcoholism, morphinism, abstinence from morphia, the auto-intoxication of diabetes, carcinoma, etc.), and *infection*. Muttering delirium is the name given to a special form, in which the patient who is possessed with sensory delusions mutters softly and unintelligibly to himself, whilst the motor restlessness is mainly limited to the hands which he moves about, as if he were trying to catch and pluck something to pieces, and so on. In other cases the delirium is associated with a strong impulse to movement, so that the patient cannot be kept in bed, tries to jump out of the window, etc.

Bleuler (*Psych. neur. Woch.*, 1902) describes a remarkable case of "unilateral delirium," which is difficult to interpret.

A not uncommon sign of organic brain disease is diminution or loss of mental power, which ranges from simple *impairment of memory* to complete *dementia*. This is due to special chronic, diffuse, and disseminated processes, which affect the cerebral cortex either directly or through the vessels.

The memory may be affected in various ways. The patient may merely have lost the power of apprehending new memory-images, or the memory of certain periods of time, such as the impressions of the immediate past, may alone be lost. Names may be chiefly or exclusively forgotten, the memory being otherwise intact, and so on. The loss of memory may extend to time and place.

Errors of memory (paramnesia) are not uncommon; the patient may think that he has already been through the same experience, etc., but this is not always a pathological condition.

Sudden loss of memory seldom follows an apoplectic attack; when this has been observed the cause has usually been syphilis (Fournier, A. Pick, Weber).

I have seen several cases of cerebral syphilis in which loss of the power of perception was the only mental symptom. In two of my cases the only other symptom was disturbance of equilibrium in walking.

Pulse, Respiration, and Temperature.—The disturbances of the action of the heart caused by organic brain disease are slowing, acceleration, and irregularity of the pulse. These appear specially in diseases of the *medulla oblongata*, or of the *vagus centre*, from some process which affects it either directly or by exaggeration of the general brain pressure. Slowing of the pulse is the characteristic symptom. In brain tumour, meningitis, abscess, etc., it may decline to 20-30 beats a minute and even less, but it seldom decreases below 44-48. This slowing may in the last stages be followed by an acceleration. Acceleration may also be the first sign of vagus affection. The rapid pulse, which as a rule accompanies fever, is not infrequently absent in brain disease; the pulse may be slow although the temperature is high. We have little definite knowledge

as to the meaning and localisation of the Stokes-Adams symptoms, viz., persistent slow pulse with epileptiform attacks.

This affection was termed by Charcot " pouls lent permanent avec attaques syncopales et épileptiformes," and was later known by Huchard as Stokes-Adams disease. We have further information on this subject from His, Webster, Kaufmann, Brissaud, Lépine, Halipré, Du Mesnil, De Rochemont (*M. m. W.*, 1903), Kidd (*Lancet*, 1904), Jaquet (*A. f. kl. Med.*, Bd. lxxii.), Laslett (*Lancet*, 1904), Gaudon (*Thèse de Paris*, 1905), Medea (*Progrès méd.*, 1905), Jacquier (*Thèse de Paris*, 1905), Lichtheim (*A. f. kl. Med.*, Bd. lxxxvi.), Schmoll (*A. f. kl. Med.*, Bd. lxxxvii.), etc. In the works of the last-named writers the heart symptoms (dissociation of the auricular and ventricular rhythm) are specially considered. A distinction is made between a *cardio-vascular* and a *neurogenic* form. The latter is observed chiefly in affections of the medulla oblongata, in compressions from tumours and affection of the bulbar vessels, but also in morbid processes of the vagus nerve. See Kraus, *D. m. W.*, 1905. Here, however, we are dealing with a group of symptoms, and not with a morbid picture *sui generis*. For that reason the course and prognosis are also different.

Moreover, we should not forget that some healthy persons have unusually slow action of the heart.

Irregularity occurs chiefly in diseases which arise from the medulla oblongata or by which the vagus centre is secondarily involved.

The *respiration* may be influenced in various ways by diseases of the brain. In coma and in increase of intracranial pressure, the breathing is usually slow and deep. This occurs also in diseases of the medulla oblongata, but these more often give rise to dyspnœa and irregular breathing. One very sharply characterised type is the *Cheyne-Stokes repsiration*, which may occur in meningitis, hæmorrhage, tumour, aneurism of the vertebral artery, etc., and in every case of coma. It has also been noted as a symptom of the disease due to a high altitude (Bergkrankheit) (Zuntz, Mosso). The respiration is of a non-rhythmic and periodic type. After a few superficial respirations the breathing becomes gradually deeper, then noisy and stertorous, and again gradually more superficial and slower. Then comes a pause, a stage of apnœa, until the cycle once more commences. Each of these stages lasts on an average the fraction of a minute, but each of them—even the pause in breathing—may be prolonged for more than a minute. During the apnœa the pupils are usually contracted and the pulse slow. The patient, who is generally dazed, sometimes regains consciousness during the stage of deep inspiration. Although the symptom as a rule lasts but for a short time, isolated cases have been noted in which it persisted for months, a year, or even longer.

Cases of this kind have been mentioned by Thomayer, Hein (*W. kl. W.*, 1877), Roth (*A. f. kl. Med.*, x.), Terrien (*Progrès méd.*, 1898), Libensky (*W. kl. R.*, 1905), etc. O. Rosenbach (in the first edition of Eulenburg's " Realenzyklopädie," 1880) had already described the condition. Terrien reports a very remarkable but unique case in which anæsthesia and loss of reflexes developed in the area of the trigeminus during the phase of respiration and disappeared during that of apnœa. Periodic changes in the size of the pupils has been often observed (Thiemich, Whitehead, Terrien, etc.). I have seen a combination of Stokes's phenomena with yawning spasm, viz., spasm in the jaw muscles and marked exaggeration of the reflex in the left nasal mucous membrane, in a coma of indefinite origin. In one case of eclampsia gravidarum a touch on the cornea during the comatose stage arrested respiration.

Traube thinks that diminished excitability of the respiratory centre is the cause of Stokes' breathing. According to Filehne the vasomotor centre plays an important part in its causation. An attempt has recently

been made, but on insufficient grounds, to trace the symptom to the cerebral cortex (Rabé,[1] Parhon-Goldstein [2]). A similar type of breathing may occur in healthy persons during sleep. It has also been observed in hysteria (Christiani, Raymond, and Janet[3]).

Ebstein (*A. f. kl. Med.*, Bd. lxxx.) states that all the transitions between Cheyne-Stokes respiration, Kussmaul's deep breathing, and simple irregularity of respiration may occur.

Biot's respiration is the name given to a rapid, short respiration, which is interrupted by sudden pauses lasting for about half a minute. There are other disorders allied to Cheyne-Stokes respiration but distinguished from it by the absence of pauses.

Kassowitz describes as another form of respiratory disorder an expiratory apnœa, and Schlesinger describes the following as an allied type: A deep inspiration is followed by rapid successive expiratory movements, with no inspiration between them; the thorax remains finally fixed in the position of expiration, and the attack closes with a deep inspiration.

L. Hofbauer[4] treats of the various forms of respiratory disorders in a monograph.

I have described a remarkable case in a lady who was suffering from the effects of severe influenza; during sleep her respiration and pulse ceased, and symptoms of asphyxia and collapse ensued. The patient had to be artificially kept awake. Simple irregularity of respiration may be caused by disease of the medulla oblongata. In rare cases the breathing has been found to cease whilst the action of the heart still continued, so that artificial respiration postponed the end (Macewen, Hoffer,[5] Fliess, etc.). The respiratory centre seems to become paralysed under the influence of increase of the brain pressure more rapidly than the heart vagus centre (Duckworth).

In diseases of the posterior cranial fossa, especially in new growths, aneurisms, etc., changes in the position of the head may produce severe disturbances of respiration and circulation (see chapter on brain tumour and aneurism of the cerebral arteries).

In infective diseases of the brain (meningitis, encephalitis, etc.), the *temperature* is as a rule raised, but in many cases this is not so. The fever of brain disease may be associated with a slow pulse. Cerebral hæmorrhage usually causes a moderate rise of temperature, but rarely high fever. The apoplectic attacks of sclerosis and paralytic dementia are usually accompanied by fever, and a steady rise of temperature is observed in status epilepticus. Any affection of the pons and medulla oblongata may produce fever, but acute and destructive processes are specially apt to do so. A rise of temperature is occasionally observed in acute diseases of the motor zone and corpus striatum, and in surgical treatment of these regions; it may under these conditions be limited to the opposite side of the body or be more marked there. A fall in the temperature is a common symptom of cerebral hæmorrhage, and it sometimes occurs in brain abscess. It is also occasionally noted in comatose conditions of other genesis.[6]

[1] *Presse méd.*, 1899. [2] *R. n.*, 1902.

[3] "Un cas de rhythme de Cheyne-Stokes dans l'hystérie," Paris, 1900.

[4] "Semiologie und Differentialdiagnostik der verschiedenen Arten der Kurzatmigkeit auf Grund der Atemkurve," Jena, 1904.

[5] *Prag. med. Woch.*, 1902.

[6] Goetze-Erdheim give some references to the literature on subnormal temperature in brain diseases (*Z. f. Heilk.*, 1905).

II. Focal Symptoms

Motor Focal Symptoms

A. Symptoms of Irritation

The fact demonstrated by Fritsch and Hitzig,[1] that stimuli which affect the motor zone of the cortex produce convulsions in the muscles of the opposite side of the body, has been confirmed and supplemented by pathology. Morbid processes which affect the motor cortical region without destroying it, give rise to tonic and clonic spasms in the muscles whose centre has been stimulated. These symptoms of motor irritation may be confined to one group of muscles or to one extremity. The spasms caused by stronger or repeated stimulations—and in disease we have usually to deal with an acute, intense stimulation, *e.g.* in hæmorrhage or trauma, or with a persistent or repeated weaker stimulation, *e.g.* in tumours—are not limited to the muscular area first involved, but extend in a regular manner over the whole of the affected side of the body, precisely as if the stimulation in the cortex had spread by contact from the centre originally affected to the others. If, for instance, the lesion is situated in the facial centre, it may at first be revealed by tremors which are confined to the facial muscles. Later, with an increasing stimulus, or one which from the first has been intense, the convulsion certainly begins in the area of the facial muscle, but subsequently spreads to the arm (and indeed usually first to the hand and fingers), then to the leg of the same side, and in the end it may involve the other side of the body also. If the convulsion commences in the muscles of the leg, it extends from these to the arm, and eventually to the muscles of the face. If the arm is first affected, the spasm usually spreads first to the face and then to the arm of the same side. There is as a rule no loss of consciousness in localised convulsions, but this may occur during the course of the seizure if the convulsions extend over the whole of one side. Loss of consciousness usually supervenes when the spasm extends to the opposite side of the body. In spreading to the opposite side it attacks first the leg or the muscles last or first involved on the side first affected. Opinions are not unanimous upon this point.

We cannot here discuss the varieties of symptoms produced in experiments on animals, nor the differences of opinion which have thus arisen between Unverricht and Prus (*W. kl. W.*, 1898). We must also omit discussion of the question of the tracts in which these stimuli are conducted, which has been studied by these writers and by Bischoff and Hering.

Those muscles which under normal conditions have a bilateral action, *e.g.* those of the trunk, jaw, larynx, palate, pharynx, and those for closing the eyes, may in unilateral spasms be active on both sides. I have repeatedly found this to be so as regards the muscles for closing of the eyes in convulsions which were otherwise strictly unilateral.

This form of localised or *unilateral spasms* is known as *partial epilepsy*, or *cortical* or *Jacksonian epilepsy*. It is caused by a condition of irritation of the motor zone, which may be either *functional* in nature (hysteria, etc.) or of *organic* cause (hæmorrhage, softening, inflammation, trauma, and very specially tumour), or may be due to *intoxication* (alcoholism,

[1] *A. f. Anat. u. Physiol.*, 1870.

uræmia, diabetes (?), lead-poisoning, etc.), and infection (and may also appear during the convalescence from feverish illnesses).

The spasms are usually followed by a condition of *transient paralysis*, which involves mainly the muscular area first and most affected by the spasm. It gives one the impression, indeed, of an exhaustion of the motor centres following on the condition of irritation, and it tends to disappear rapidly.

There may also be attacks of paralysis not due to the spasms, but apparently of primary origin. These last only for a short time. Higier (*Z. f. N.*, xiv.) regards them as equivalent to cortico-epileptic attacks, referring in so doing to the theory of Hering and Sherrington of the inhibitory function of the motor cortical region.

The nature of the causal process suggests, however, that the cortical centres are not only irritated but are more or less destroyed. This explains why permanent paralytic conditions are often associated with cortical epilepsy, and acute, destructive affections (hæmorrhage, softening, etc.) are from the first combined with spasms, the onset of which they accompany or precede, whilst chronic, gradually-increasing, morbid processes—new growths in particular—are for a long time revealed solely by the convulsive symptoms, the paralysis following gradually or in successive attacks. The lesion which causes the spasm need not directly affect the motor cortex, but may merely lie so close to it as to produce in it a condition of irritation. The lesion which gives rise to *paralysis* of the cortical centres must be situated in these centres themselves, or must inhibit their function by pressure.

If the spasm is at first local, and only extends during later attacks, this, in addition to a similar development of the paralysis, points to a slow, progressive process, and shows that the cause is undoubedly an organic disease.

An obvious pathological change is very rarely absent under such conditions (see remarks on so-called pseudo-cerebral tumour in the section on brain tumour).

True epilepsy is seldom ushered in by localised muscular twitchings, and is also seldom limited to one side of the body, but the observations of Loewenfeld,[1] Oppenheim, Binswanger,[2] Reynolds,[3] L. Müller,[4] and others prove beyond doubt that this variety does occur. It should be further mentioned that general convulsions, which cannot be distinguished from ordinary epilepsy, sometimes take place in diseases of the motor zone.

Partial epilepsy may also affect the *sensory* area. Paræsthesiæ in one limb or part of a limb may usher in the spasmodic attack; they may accompany the tremors or may be the only symptom of the irritative condition, and thus represent to a certain extent an equivalent of the attack. The importance of these attacks as regards local diagnosis is not yet determined, but they certainly occur in diseases of the motor region of the brain or of the posterior central convolution. Tachycardia has also been occasionally noted in cortical epilepsy.

I have observed a group of symptoms, viz., attacks of cyanosis in one arm and the face on the same side, with or without loss of consciousness, which seemed to form a vasomotor variety of

[1] *A. f. P.*, xxi. [2] Nothnagel's "Handbuch," xii. [3] *Brit. Med. Journ.*, 1899.
[4] *Z. f. N.*, xxviii. See here the literature on partial epilepsy without anatomical changes. Also Béla Konrad, "Gyógyaszat," 1905.

Jacksonian epilepsy. These symptoms finally became persistent and were associated with myosis and narrowing of the palpebral fissure, and eventually with a paresis in the arm and face.

Unilateral contractions may also be produced by diseases of *the subcortical* white substance, especially if these affect the nerve tract coming from the motor zone. They do not, however, fully correspond to the type of Jacksonian epilepsy, and when they do resemble it, they are due to affections which interfere with the cortex itself, by pressure, by a distant, or by a reflex action. Thus, as Weber[1] has specially shown, even cerebellar foci may give rise to convulsions affecting the whole of the same side, a symptom which, however, I have only observed in tumours. Cortical epilepsy disappears when the cortical centres are completely destroyed.

Diseases which lead to an increase of intracranial pressure may give rise to convulsions at any site. These spasms, due to general increase of intracranial pressure, are *general*, and correspond more or less completely to *true epilepsy*.

Clonic contractions which persist for days and weeks are very seldom produced by disease of the motor centres. Persistent clonic facial spasm has, however, been observd in the region of the facial centre in tumour. I have seen a steadily repeated rhythmic contraction in the muscles of the toe persist for days in a tumour of the leg centre and in a meningo-encephalitis of this region, a clonic contraction which continued for hours and days, although with interruptions, in the right arm, the right side of the face, and in the muscles which move the eyeballs towards the right, in a tumour of the left frontal lobe and of the motor region, etc. etc. This form of spasm also occurs in paralytic dementia. A true " status hemiepilepticus " may develop from an accumulation of attacks of this Jacksonian type, and this form cannot always be identified with pathological change. Toxic epilepsy (alcoholism) in particular may assume this guise. Cases of this kind have been described by Hitzig, Gowers, Winkler,[2] and especially by L. Müller.[3]

Kemmler (" Arbeit. aus der psychiatr. Klinik," Breslau, 1895) describes spasmodic attacks with rhythmic contractions synchronous with the pulse. O. Fischer has shown, however, that the rhythmic contractions are not synchronous with the pulse (*M. f. P.*, xxi.).

It is not yet certain whether *choreic tremors*, *athetosis*, and allied symptoms of motor irritation (see below) may arise from the cortex. Choreic-athetotic movements have repeatedly been observed in tuberculous meningitis and especially in the form which develops as localised meningo-encephalitis (méningite en plaques tuberculeuse) chiefly in the zone of the fissure of Rolando (Boinet, Boncarut). A case described by Chipault also points to the possibility of a cortical origin of hemichorea. We shall, however, refer to this symptom in connection with hemiplegia, to which it is closely related.

Tetanic spasms have been observed in many cases of affection of the cerebellum, and especially in tumours. These are attacks of tetanic rigidity of the muscles of the whole body, with opisthotonus, resembling those in tetanus. This tonic contraction is, however, usually interrupted by single jerks—as if an electric shock passed through the whole body—or by general clonic contractions. This condition, and the peculiar

[1] *M. f. P.*, xix. [2] " Congrès internat. de Psych., etc., de Bruxelles," 1897.
[3] *Z. f. N.*, xxviii. ; see also Bonhöffer, *B. k. W.*, 1906, and Auerbach and Grossmann, *M. m. W.*, 1907.

attitude caused by the tonic muscular contraction allied to it, were first described by Hughlings Jackson.[1] It appears that these and kindred spasmodic conditions may be produced in sucklings by reflex, and specially by toxic causes (from the intestine).

I [2] have seen persistent contractions of the laryngeal muscles and of the soft palate in tumour of the cerebellum which pressed upon the medulla oblongata. Cases of this kind are also described by Klien,[3] Sinnhuber,[4] and others.

B. Paralysis

Destructive diseases of the motor centres and conducting tracts form the basis of the paralysis which develops in brain diseases. Paralysis of *cortical* origin is distinguished from that due to affection of the motor tracts by the fact that it is confined as a rule in the form of *monoplegia*, to single segments of one side of the body. This is not surprising if we consider that the motor centres extend over a large area of the cortex, whilst organic brain diseases usually assume the form of circumscribed foci. The blood supply of this area is also distributed among various arteries. The monoplegia is, however, more often a paresis than a complete paralysis.

A disease of the cerebral cortex may thus be limited to the facial centre and may manifest itself by a *facial monoplegia*. If the lowest zone of the anterior central convolution is affected to a somewhat greater extent, a *facio-lingual monoplegia* will develop. It much more often happens, however, that the process involves the arm centre and partially or entirely inhibits its function, so that symptoms of a *facio-brachial monoplegia* arise. Or in addition to the facial, some muscles of the arm, usually those of the hand and fingers, are paralysed. The paresis may even attack the thumb alone, or the fingers without the thumb. Lesion of the cortical centres may be revealed by the fact that the grasp of the fingers can no longer be properly carried out, that the power of isolated movements of the fingers is lost, and, as Marinesco has specially described, troublesome associated reflex movements are thus produced, (*Semaine méd.*, 1903). Partial paralysis of the area supplied by the musculo-spiral nerve, of cortical origin, has been described by Pick and myself. If the lesion is situated exclusively in the paracentral lobes or the uppermost summit of the central convolutions, the clinical symptoms may be those of a pure *crural monoplegia*. There may even be a cortical paralysis confined to the area of the peroneus or even to the extensor hallucis longus.

Brachial monoplegia is caused by a lesion limited to the middle third of the anterior central convolution. If the upper two-thirds of the central convolutions or the anterior central convolution are alone affected, the arm and leg are paralysed and the cranial nerves (vii. and xii.) are spared. Finally, widespread lesion of the whole motor zone gives rise to hemiplegia.

Monoplegia is characteristic of cortical affections of the motor region; it is very seldom caused by diseases of the medullary substance. On

[1] *Brit. Med. Journ.*, 1871. See also Horsley, *Br.*, 1906; Dana, *New York Med. Journ.*, 1903; Foerster, "Die Kontrakturen," etc., Berlin, 1906; Ziehen, *Med. Klinik*, 1905, etc.
[2] *N. C.*, 1889. [3] *D. m. W.*, 1904. [4] *B. k. W.*, 1904.

the other hand the results of brain surgery show that superficial cortical lesions which do not penetrate into the medulla, do not produce persistent paralysis. Subcortical foci lying immediately below the grey matter of the motor cortex may give rise to monoplegia. The more deeply the lesion is situated within the centrum ovale, the greater is the number of motor fibres which it destroys; it usually then produces that form of paralysis which corresponds to the interruption of all the motor nerve tracts, viz., *hemiplegia*. Paralysis of a monoplegic character has, however, been observed in isolated cases of affection of the internal capsule. It seems to me that hemiplegia is more easily produced by diseases of the cortex in children than in adults.

Paralysis of the muscles of mastication, deglutition, and of the larynx is hardly ever produced by unilateral cortical foci, or only under special conditions, but on the other hand bilateral lesions of the corresponding centres are capable of causing a bilateral paralysis of these muscles.

Cortical epilepsy and *monoplegia* are the attributes of cortical disease in the motor central region. The former is the symptom of irritation, the latter shows the loss of function which may be caused by inhibition, intoxication, or destruction (hæmorrhage, softening, inflammation, abscess, etc.).

Monoplegia is usually associated with *exaggeration of the tendon reflexes* and hypertonia, but hypotonia also occurs in exceptional cases. The conditions to which these different symptoms are due have not yet been sufficiently investigated.

The muscles which cannot be voluntarily moved may enter into action under other conditions, *e.g.* in emotion, or in gesticulation, from reflex influences or in associated movements. Thus I have treated a woman who had suffered for a long time from a cortical monoplegia of the arm due to tumour of the motor zone, and who could not move the arm in the slightest degree; when, however, she became excited during the beginning of chloroform anæsthesia, strong movements of self-protection appeared in the paralysed extremity. Moreover, cortical lesions of the motor cortical centres appear permanently to affect only the principal movements (see footnote on p. 623), whilst the so-called common movements may be retained.

The term "mind-paralysis" (Seelenlähmung) has been used by various writers (Munk,[1] Krafft-Ebing, Nothnagel,[2] Bruns [3]) in different senses. By it Nothnagel understands the loss of the memory of motor images for the extremity or one side of the body. It would thus practically coincide with Meynert's motor *asymboly*, except that he localises the lesion in the central area, whilst Nothnagel localises it in the parietal lobe. On the other hand the terms *asymboly* (Finkelnburg), *agnosia* (Freud, Claparède), have been applied to conditions in which there is loss of comprehension of the conventional signs or symbols. Bruns [3] has a different conception of "mind-paralysis"; he describes conditions of apparent paralysis of one side of the body, in which there seems at first to be no power of active movement, but in which the patient

[1] "Über die Funktionen des Grosshirns," 2nd ed.
[2] "Verhandl. d. Wiesbadener Kongresses," 1887.
[3] "Über Seelenlähmung." Festschr. zum 50 jährigen Bestehen von Nietleben, 1897, and *N. C.*, 1898.

can use the muscles by making a special effort, and may even in the end be able to move them with normal power. Bruns attributes the symptom to the fact that the sensory stimuli which initiate the movement no longer flow to the corresponding motor centre. The muscles can, it is true, still be voluntarily moved, but the limb is to a certain extent lost to the patient because the association tracts by which the proper motor centre is excited to action are injured or cut off. I have observed a somewhat similar condition in affections of the parietal lobe.

The term "*apraxia*" had also been used even earlier, *e.g.* by Kussmaul and Griesinger, partly in the sense of asymboly, and partly to indicate the loss of certain combined motor acts with the preservation of the elementary movements in the same muscle groups, as for example loss of power to protrude the tongue, although its motility is intact, confusion of the gestures of affirmation and negation, etc. Lewandowsky speaks of an apraxia of closing the eye.

Liepmann,[1] however, has done us great service in the elucidation of this question, and by his masterly analysis he has thrown open to us this dark region of brain pathology.

He has applied this name to a functional affection which is related to mind paralysis and aphasia, and is characterised by the fact that the muscles—*e.g.* of one limb or one side of the body—while they can be moved, cannot be moved to any purpose, or used for any suitable action—a symptom which is not due to inco-ordination or to any loss of the proper conception of the object in view. By the word action he understands all the acquired combinations of elementary muscular actions which either represent operations upon the objective world—knocking, ringing, sealing, lighting a cigar, etc.—or reveal the mental processes to others by the movements of expression. The patient makes awkward movements with the apraxic limb which do not correspond to his object; *e.g.* he puts his tooth-brush in his mouth instead of his cigar; he cannot threaten, sign, or salute with the affected extremity, etc. Liepmann attributed the disorder to the fact that the sensori-motor centre of the affected limb is in itself intact, but is cut off from its connection with the rest of the cerebral cortex. A disease of the parietal lobe, as the result of which the motor zone is cut off from the temporal and occipital lobes, and a simultaneous destruction of the corpus callosum which severs the connection with the other hemisphere, seemed to him best suited to explain the functional disease. Later he laid stress chiefly on the lesion of the corpus callosum, as he[2] had succeeded in demonstrating the interesting facts that the left hemisphere predominates over the right in regard to actions, and that the sensori-motor centres of the right hemisphere are to a certain extent dependent upon the left and under their control, A more or less marked apraxia or dyspraxia of the left hand is therefore frequently associated with right cortical and subcortical, though not with capsular, hemiplegia (see following section) and aphasia.

This apraxia of the left hand may also be produced: 1. by cortical and subcortical foci which destroy the sensori-motor centres of the left hemisphere and the fibres which it sends by the corpus

[1] *M. f. P.*, viii.; *N. C.*, 1904; also "Über Störungen des Handelns bei Gehirnkranken," Berlin, 1905; see further *M. f. P.*, xvii., and "Der weitere Krankheitsverlauf bei dem einseitig Apraktischen," etc., Berlin, 1906, and *M. f. P.*, xix.; *N. C.*, 1907.

[2] Liepmann, *M. m. W.*, 1905; *N. C.*, 1905; *Med. Klinik*, 1907.

callosum to the corresponding centres of the right hemisphere. In this case apraxia of the left hand is associated with paralysis of the right. 2. By lesion of the corresponding callosal fibres in the left, and 3. by interruption within the right hemisphere, before their entrance into the cortex, of the callosal fibres which stream from the left into the right sensori-motor centre.

Liepmann further arrives at the conclusion that the sensori-motor centres contain merely the memory images and the directive functions for simple movements and the common simple motor acts, whilst actions, complicated purposive movements, are related to the co-operation of various zones of the brain, and especially to the left hemisphere. Although at first he also ascribed some importance to the parietal lobe in so far that its destruction is specially likely to sever the sensori-motor region from the rest of the brain, especially from the occipital region, he also on the other hand considered the possibility that the left frontal region might play a prominent part in actions. Hartmann [1] had stated this point still more definitely. He thought the higher co-ordination of simple motor acts into actions was related to the frontal lobes, especially to the left. He also recognised that interruption or lesion of the corpus callosum might cause apraxia (conduction-apraxia) by depriving the right hemisphere of its control by the left frontal lobes, and also because the corpus callosum effects purposive co-ordination of the two upper extremities in actions. He

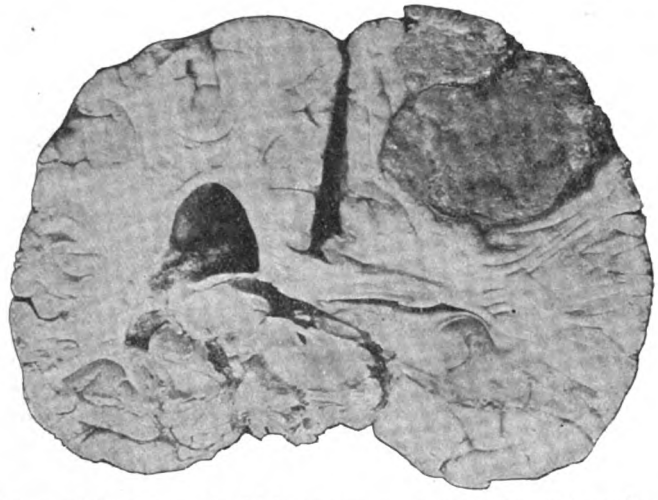

FIG. 296.—Tumour of the right parietal lobe, præcuneus, compression and atrophy of the corpus callosum with apraxia of the left arm. (Oppenheim.)

admits the theory of Ramón y Cajal that the left hemisphere contains unilateral attention centres —memory centres—in addition to the bilateral projection centres, but he by no means goes so far as to say that the right frontal lobe is of no importance whatever in this respect.

I have had a case in which a tumour of the right superior parietal lobe, involving the corpus callosum, had caused apraxia of the left hand (Fig. 296), and I have also seen apraxia of the left hand follow removal of a tumour of the left parietal lobe.

Heilbronner [2] and Pick [3] have published further studies on apraxia. The former is inclined to speak of a cortical apraxia, in the sense of Nothnagel's mind paralysis, and to contrast it with Liepmann's apraxia as a transcortical affection. Pick has gone still further in the differentiation of the various forms; he has also put forward the conception of "ideatory" apraxia, and has pointed out the great rôle which psychic factors, such as disorders of the attention, may play in the genesis of these symptoms, especially emphasising the importance of perseveration (power of continuance).

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[1] *M. f. P.*, xxi. [2] *Z. f. Psychol.*, 1905.

[3] "Studien über motorische Apraxie," etc., Leipzig, 1905. See also Kleist, *M. f. P.*, xix., and *Jahrb. f. P.*, Bd. xxviii.; Margulies, *M. m. W.*, 1907; K. Abraham, Westphal, *Z. f. Psych.*, Bd. lxiv.; also Maass, *N. C.*, 1907; Roose, "L'Encéphale," 1907; Monakow, *Ergebn. d. Physiol.*, vi., 1907, whose views cannot be further considered here. See also the discussion at the Amsterdamer internat. Kongress, 1907, ref. *N. C.*, 1907.

All the motor nerve tracts of the opposite side of the body are contained in the *internal capsule*, and indeed in its posterior limb. Lesions of this region almost always give rise to *hemiplegia*, *i.e.* paralysis of the leg, arm, facial muscles, and tongue. In the face the paralysis is, as a rule, limited to the *inferior facial region*. The mouth is drawn towards the unaffected side and can only be moved on this side. The asymmetry may disappear under certain conditions in mimic movements, especially in laughing. The superior facial is intact, or is affected only to such a degree that the eye cannot be closed so tightly as on the sound side. The patient can usually only contract the orbicularis of the paralysed side in common with that of the sound one. It should be remembered, however, that many persons can only contract both the orbicularis muscles at the same time (bilaterally), and that those occupied in shooting, drawing, microscope work, etc., acquire the power of closing one eye on one side only. Further, the patient cannot keep the eye closed so long as on the unaffected side (Saenger). The eyebrow is sometimes lower, and the frontalis does not contract with its full strength. There is even, in very rare cases, a slight lagophthalmus.

FIG. 297.—A case of left hemiplegia. Deviation of the outstretched tongue towards the left side. (Oppenheim.)

Mirallié, Féré, and Saenger (*N. C.*, 1899) have recently pointed with much emphasis to the involvement of the superior facial in hemiplegia, a fact which has been known for a long time and which was described in the earlier editions of this work. There is, however, no doubt that while the paralysis of the inferior branch may be complete, the superior branch may be intact, and that the disturbance of innervation in the muscles of the superior facial is as a rule very slight. On the other hand it seems to me from my own observation that there is often very marked paresis of the superior facial in facio-brachial monoplegia, when the lesion is in the cortex.

An involvement of the *hypoglossus* is indicated when the protruded tongue deviates towards the paralysed side (Fig. 297). This symptom is due to the fact that the genioglossus is chiefly concerned in this motor act, and by its contraction it moves the tongue towards the opposite side.

Beevor (*Br.*, 1906) has recently made an experimental study of this question.

Unilateral paralysis of the facial and hypoglossal often causes slight affection of the articulation, but as a rule it is of short duration.

If the hemiplegia is incomplete, the hand and fingers, the foot and toes are usually more severely affected than the other muscles of the extremities. Even when the hemiplegia has developed in the typical manner, the paralysis is not all the time equally distributed over all

the muscles of the extremities; the extensors of the foot and flexors of the knee are usually most affected and as a rule they remain permanently paralysed (Wernicke [1]). The muscles which open the hand and rotate the extremity outwards tend to remain paralysed (Mann [2]).

The muscles of *mastication* [3] *and deglutition*, those of the *larynx* and usually those of the *trunk*—which are all normally *bilateral* in their function—are quite intact. It is assumed that each hemisphere contains centres for the muscles of both sides of the body which have a bilateral action, so that paralysis of one side does not produce a marked defect. This explains the slight involvement of the muscles for closing the eyes. It should also be remembered that the so-called common movements (Munk [4]) are specially controlled by subcortical centres and that they probably receive the impulse for activity from various cortical zones. This is probably true also as regards the *ocular muscles*, which are very rarely, if ever, involved in simple hemiplegia.[5] The deviation of the eyes and head towards the unaffected side, which sometimes accompanies hemiplegia, is a symptom of transient duration, which will be discussed later.

Of the trunk muscles, the trapezius is most often affected; the shoulder of the paralysed side cannot be raised at all or not so well as that of the other side, whilst the sterno-mastoid is contracted in the normal way. In deep breathing the thorax may be less well expanded on the paralysed than on the healthy side (Nothnagel).

The investigations of Boeri and Simonelli (*Gaz. degli Osped.*, xxi.) show that this is no uncommon symptom. Jackson observed that in quiet breathing the superior intercostal muscles contract more on the paralysed than on the unaffected side, but on the other hand they contract less in deep breathing. This is confirmed by Clark (*Amer. Journ. Med. Sc.*, 1903), Judson Bury (*Lancet*, 1903), and Weisenburg. Severe paralysis of the respiratory muscles has been rarely observed (Bonhoeffer). Sicard mentions involvement of the abdominal muscles (*Arch. de Neurol.*, 1899).

In recent hemiplegia the leg lies upon the bed, like that of a cadaver, the thigh being broadened or flattened and shortened in its long diameter. Heilbronner (*Z. f. N.*, xxviii.) regards this sign of the "flattened leg" as an important symptom in hemiplegia.

In bilateral foci, paralysis may also develop in the muscles of deglutition, mastication, and of the larynx (see chapter on pseudo-bulbar paralysis). Unilateral foci seldom have this effect. In such cases there is probably an individual peculiarity, the corresponding centres of one hemisphere having a marked preponderance. The muscles of the neck and trunk have been affected in a few cases of this kind, and they are almost always involved in congenital diplegia (*q.v.*).

A so-called *homolateral* or *collateral* hemiplegia, in which there is unilateral paralysis on the side of the lesion, is very rarely observed. In the cases described under this diagnosis, there has usually been some error of observation, lesions in the opposite hemisphere or in the pons and medulla oblongata (hæmorrhage, œdema, softening, etc.), which have implicated the motor pyramidal tracts, having been overlooked on account

[1] *B. k. W.*, 1889, and *D. m. W.*, 1895. [2] *Z. f. N.*, x.; *M. f. P.*, iv.

[3] The divergent views of Mirallié-Gendron (*Rev. de Path. nerv.*, 1906) are hardly calculated to modify this teaching.

[4] *Sitzungsb. d. K. pr. Ak. d. W.*, 1892-96.

[5] Compare also Desclaux, *Thèse de Paris*, 1903; Mirallié, *R. n.*, 1903, 1904; and Wilson, *R. n.*, 1904. These writers maintain that by testing with a prism one can discover a latent oculo-motor paresis in recent hemiplegia. But this symptom is of no real importance.

of their insignificance. Moreover processes arising from one hemisphere, especially tumours, may press so much towards the other hemisphere that it suffers chiefly from the compression. The most common cause of error is, however, that flaccidity and immobility of the limbs of one side occurring in coma is wrongly diagnosed as hemiplegia, and the symptoms of motor irritation on the other side are mistaken for signs of activity (Pineles, Ortner). In the few cases which cannot be explained in this way, the cause has been assumed to be some defect of development, such as a congenital failure of the decussation of the pyramids; but this has only been demonstrated in a few cases (by Pitres,[1] Zenner, Dupré, Camus,[2] and others). This collateral hemiplegia has repeatedly led to errors in surgical treatment.

Spielmeyer (*M. m. W.*, 1906) concludes from one case that a hemiplegia may in exceptional cases develop from a diffuse process in the cerebrum if the pyramidal tract is intact. He assumes that it is due to isolation of the motor neurones by the diffuse disease.

The hemiplegia, even when it is a *direct focal symptom*, subsequently undergoes certain modifications, *i.e.* when it is caused by destruction of the motor-nerve tract. Power of movement returns to some of the paralysed muscles. Paralysis of the tongue may rapidly disappear, but recovery is less often the case as regards the paralysis of the face. In almost every case the leg regains the power of movement so far that the patient is once more able to walk, but it remains so feeble that he drags it along. The extensors of the foot and toes generally remain permanently and completely paralysed, and the patient is compelled, by the equino-varus position of the foot, to drag the leg, the toes of which rest on the ground, round in a semicircle to the front of the other leg. In walking sideways the patient can more easily move towards the affected than towards the unaffected side (Schüller,[3] with whom Campbell and Crouzon agree). The flexors of the knee usually also remain paralysed. The power of movement returns to the arm later and less completely than to the leg. The patient generally learns to move the arm slightly in the shoulder- and elbow-joints, whilst the hand and fingers remain entirely, or almost entirely paralysed.

This return of motility may be due to the fact that some of the pyramidal fibres have regained their power of function, or possibly, although they are completely destroyed, the subcortical centres, the thalamus, the extra-pyramidal tracts in particular (see p. 637), and the motor bundle of the tegmentum, have become to some degree compensatory for them. This theory has been specially elaborated by Rothmann (*M. f. P.*, xvi.). It would explain the restitution of the coarse common movements and of the bilateral symmetrical movements, especially as these are controlled by either hemisphere. Rothmann thinks, however, that the unaffected hemisphere may, with time, create paths for the restitution of isolated movements in the paralysed limbs, after the subcortical centres of the affected hemisphere have acquired a certain independence in their function.

Another symptom which accompanies the hemiplegia when it is a direct focal symptom, is *contracture*. Two forms of contracture are recognised, an early and a late. The former may develop within the first few hours or days and may again rapidly disappear. It is probably caused by irritation of the pyramidal fibres. It has been specially observed in cases of hæmorrhage into the ventricles. According to Foerster it is characterised by contraction of all the muscles of the

[1] "Les centres moteurs corticaux," etc., Paris, 1895.
[2] *R. n.*, 1905.
[3] *N. C.*, 1903.

extremity or side of the body (agonists and antagonists). This often makes it difficult to elicit the tendon reflexes.

Late contracture only appears in the course of some weeks or later, seldom before the end of the second week (according to Dejerine usually between the sixth and twelfth weeks). It is a permanent symptom which certainly goes hand in hand with the descending degeneration of the motor nerve tracts, but is not due to it. The arm is usually in the following position: the upper arm is adducted, the forearm bent at a right or acute angle, the hand flexed and pronated, the fingers flexed either in all the joints or in the interphalangeal joints only. A contracture of the arm in the position of extension is rare. When the contracture is slight, the fingers can be extended. The leg is in the position of extension, the foot usually in the equino-varus position. Flexion contracture of the legs is very uncommon (cases of Prochátzka,[1] Devic-Gallavardin,[2] etc.), and occurs only under unusual conditions. The contracture seldom extends to the facial, and most rarely of all to the hypoglossus (this is quite exceptional in our experience), so that the protruded tongue in the later stages deviates towards the unaffected side (Minor[3]). The contracture is due to persistent contraction of the muscles, which disappears during sleep and tends to be less marked on waking in the morning. It can be passively overcome, but only by the use of force, and it at once returns. The tension can only be easily relaxed if the points of insertion of the contracted muscles are approached to each other; thus the fingers can be extended when the wrist is passively flexed. It is also sometimes possible to extend the fingers when the hand is supinated and the upper arm rotated outwards (Ghilarducci). The contracture is increased by any sensory stimulation, especially by cold. Artificially produced anæmia of the extremities by means of an Esmarch's bandage has a relaxing effect upon the contracted muscles (Brissaud). The contracture is combined with *exaggeration of the tendon reflexes*, in the arm as well as in the leg. An increase of the tendon jerks, which sometimes appears even within the first hours after the onset of the hemiplegia, is no indication, however, that a contracture will follow.

Of the numerous theories as regards the *contracture*, only a few can be discussed here. Charcot thought it depended upon the secondary degeneration, as he assumed that this gave rise to a condition of irritation in the anterior horn cells—by an action analogous to that of strychnine. Hitzig regarded it as related to associated movements (see below). He has since somewhat modified his theory. Hering (*A. f. Phys.*, Bd. lxx.), on the other hand, states that the unequal distribution of the paralysis is merely an apparent one, and is due to physiological causes. Dejerine-Crocq, Redlich (*Jahrb. f. P.*, 1902), and Marinesco (*Sem. méd.*, 1898), have expressed similar views, and Rothmann (*M. f. P.*, xvi.) especially maintains this view and brings out the connection between the symptom of the physiological preponderance of the extensors or elongating muscles of the leg over the flexors and the erect attitude in man. In the gradual recovery from hemiplegia, all the impulses from the subcortical centres find their way first into those groups of muscles, and thus the corresponding anterior horn-cells acquire a condition of irritation, which finds its expression in the contracture. Weisenburg (*Univ. of Penn.*, 1905) holds this view. Another theory is held specially by Gehuchten (*Journ. de Neurol.*, 1896 and 1897) in agreement with Jackson and Bastian, viz., that the pyramidal tract has a lowering influence upon the muscle tonus (and on the tendon reflexes), whilst the cortico-ponto-cerebello-spinal tracts passing from the cerebellum into the spinal cord convey impulses to the muscles which increase their tonus. If the lesion is limited to

[1] "Casop. lék.," 1902, ref. *N. C.*, 1903.
[2] *R. n.*, 1903.
[3] *Festschrift f. Leyden*, 1902.

the pyramidal fibres, whilst the cerebello-spinal tracts remain intact, hypertonus will appear, etc. There is, however, no complete parallelism between the tonus and the condition of the tendon reflexes.

Rothmann (*N. C.*, 1904) opposes this theory of the influence of the cerebellum and of the inhibitory impulses, whilst Lewandowsky's views resemble those of Gehuchten in many points We can only refer to the work of Lazarus (*Z. f. physik.*, Th. v. and vi., etc.), which contains much that is Utopian and open to dispute, and to the more recent work of Lewandowsky (*Z. f. N.*, xxix.) and of Crocq (*Journ. de Neurol.*, 1901).

None of these theories are so satisfactory as the one which Monakow puts forward in accordance with the view of Hitzig, viz., that the contracture is produced by the influence of the sensory impulses upon the lower motor system, which becomes still more important when the voluntary influences or those of the cortex are suppressed. Monakow has recently supplemented and modified this view by his theory of diaschisis.

Foerster ("Die Kontrakturen bei den Erkrankungen der Pyramidenbahn," Berlin, 1906) speaks of "paralytic contractures," and attributes them on the one hand to the loss of the inhibiting influences which are conveyed by the pyramidal tract, and on the other hand to the fact that each muscle group adapts itself to the approximating of its points of insertion by the gradual development of contraction and permanently retains this condition of shortening. This disposition is proper to all the muscles, though in varying degree, but it develops only after pyramidal conduction is suppressed. It is, therefore, one of Munk's "symptoms of isolation." The accidental position of the limbs therefore plays an essential part as regards the form of the contracture.

Hypotonia is rare in hemiplegia; we do not yet know the conditions which cause it. I have occasionally found it in cases of total hemiplegia with hemianæsthesia, etc., due to extensive destruction of large areas of the hemispheres, including the subcortical ganglia. It may be present, naturally, when the hemiplegia is caused by some disease which is associated with marked increase of the intracranial pressure (cerebral tumour). A few cases seem to show that lesions of the motor bundle of the tegmentum may be associated with diminution of the muscle tonus (Wallenberg, Halben-Infeld). Gehuchten localises the tract for the tendon reflexes in this bundle.

The *condition of the tendon reflexes* is inconstant in hemiplegia. All that we can definitely say is that they are usually increased after the comatose stage is past and that they are always exaggerated in the stage of contracture. Thus Ganault (*Thèse de Paris*, 1898), in his investigations on a great number of cases, found exaggeration in 92 per cent. of the whole. I believe that when they are not increased in severe hemiplegia, a complication is as a rule present. This has led me in several cases to suspect the existence of tabes, which was confirmed by further investigation. I have noted this also in brain tumour (*q.v.*). *Babinski's sign* is often present, but in my experience it is by no means constant. Ganault found it in 85 per cent. of his cases, Proházka ("Casop. ces. lek.," 1902) in 65 per cent. Goldflam (*N. C.*, 1903) and Gräffner (*M. m. W.*, 1906) found similar results. It may be lesion of the central ganglia, of the superior peduncles, of the cerebellum, and of the motor tracts of the tegmentum which causes unilateral motor symptoms with a normal toe-reflex (Bonhöffer, Homburger,[1] Halban and Infeld,[2] Oppenheim) and normal or even diminished muscle tonus, but these relations are by no means clearly explained. The *dorsal leg sign* (Oppenheim) is also frequent but not constant. Both these reflexes are of great value, however, from the fact that, even in apoplectic coma, as well as in very slight attacks and very transient paralysis, they indicate the pathological condition and the site of the lesion. The Bechterew-Mendel dorsal foot reflex is also often present.

The *cremasteric* and *abdominal reflexes* are as a rule absent or diminished on the paralysed side (Jastrowitz,[3] Rosenbach[4]). In my experience, this is the rule in ordinary hemiplegia. Gehuchten and Crocq speak of the antagonism or dissociation of the cutaneous and tendon reflexes, but this applies only to the above-named cutaneous reflexes. Redlich (*N. C.*, 1905) states that there may in exceptional cases be exaggeration of the abdominal and cremasteric reflexes in hemiplegia, which he attributes to conditions of irritation in the corresponding areas of the cortex.

There is practically always exaggeration of the supinator and triceps jerks in the stage of contracture, and this usually persists for a long time, even in slight and transient hemiplegia. Naturally when there is an increase of the muscle tonus, muscular contractions may often also be elicited from other points, such as the ulnar styloid process, the metacarpal bones, etc., but it would be

[1] *N. C.*, 1903. [2] *Obersteiner*, ix. [3] *B. k. W.*, 1875. [4] *A. f. P.*, vi.

idle to regard each of these symptoms as a special phenomenon. There is less frequently a wrist clonus on abrupt extension of the flexed hand or fingers, and a clonus may sometimes be elicited by sudden supination.

I have noticed that a movement of pronation (pronator sign) sometimes occurs when a fold of skin is pinched on the inner side of the forearm, or when it is stroked with the handle of the percussion hammer, but this sign is quite inconstant. Internal rotation of the arm or flexion of the hand and fingers is less often produced in this way.

The muscles of the paralysed side of the body are not materially wasted, and, if we except slight changes in the form of exaggeration and diminution, they react normally to electricity. It is only after a long time that the muscles slightly decrease in size owing to their inactivity.

Personal experience leads me to doubt the statements of Marinesco (*Semaine méd.*, 1898) and his pupils (Parhon-Popesco, etc.), as regards degenerative changes in the muscles, and of De Grazia as to changes of the electrical excitability in hemiplegia.

In a few cases, however (Quincke,[1] Eisenlohr, Borgherini,[2] Steinert,[3] Oppenheim, and others) marked and early atrophy which was not due to inactivity developed even in the limbs which had regained their motility; this was associated with a more or less marked *diminution of electrical excitability*; slight qualitative changes were even found by Eisenlohr and by myself in one case, but there was never any marked reaction of degeneration. The view that the descending degeneration of the pyramidal tract has in such cases spread to the anterior horns (as Charcot thought[4]) cannot be applied in every instance (Senator,[5] etc.), and for the present therefore we have no satisfactory explanation of the atrophy which in *rare cases* accompanies cerebral monoplegia and hemiplegia.

I find it very difficult to understand how Steinert has been led to affirm that atrophy is a constant symptom in hemiplegia (and in all supranuclear paralysis). His statement that myasthenic reaction is present also urgently demands re-examination. He holds the absence of stimuli responsible for the development of the atrophy. Weisenburg (*Journ. Amer. Med. Assoc.*, 1905) also notes the frequency of this symptom.

I have sometimes seen a rapid development of atrophy after a surgical operation in the motor region. The assumption that the brain contains trophic centres for the muscles in the motor region and in the central ganglia (Quincke) has no sufficient foundation. If this were so the infrequent occurrence of this symptom would be a very remarkable fact. Monakow (with whom Chatin[6] agrees) has also opposed this theory. He is of opinion that the deficiency of sensory, motor, and vaso-motor impulses may be the cause of the muscular atrophy. *Affections of the joints* are often present, and an attempt has been made to regard this muscular atrophy as secondary and arthritic (Darkschewitsch,[7] Gilles de la Tourette). Quincke opposes this assumption. There is no doubt that an atrophy of neuritic origin may develop in one of the affected limbs. Vasomotor affections have been held responsible by Roth, Monakow, and Bechterew,[8] and this accords with my own experience. In the cases under my observation in which the atrophy was very pronounced, cyanosis was also present, and, from the description of the patients, had been preceded by œdematous swelling.

[1] *A. f. kl. M.*, Bd. xlii.; *Z. f. N.*, iv.
[2] *A. f. kl. M.*, Bd. xlv., and *Riv. sper.*, 1890.
[3] *Z. f. N.*, xxiv. (with literature), and *A. f. kl. M.*, Bd. lxxxv.
[4] "Leçons sur la localisation," Œuvres Compl., iv.
[5] *B. k. W.*, 1879. [6] *Rev. de Méd.*, 1900. [7] *A. f. P.*, xxiv. [8] *Z. f. N.*, xvii.

It is already well known, and has recently been emphasised by Parhon-Goldstein,[1] that hemiplegia is often preceded by vasomotor disorders. In a very small number of cases of hemiplegia, atrophy of the bones has been observed on the paralysed side (Dejerine-Théohari). For details as to the vasomotor-trophic symptoms, see the following section.

We have still to mention that the muscles of the unaffected side of the body often, perhaps always, lose a certain degree of power. This is often very obvious in the leg, and exaggeration of the tendon reflexes is also frequently present in the unaffected side. I must maintain this fact in opposition to Marie-Guillain (*R. n.*, 1904), although it must be admitted that these symptoms are not of marked intensity in the healthy side. In a monoplegia of childhood, I have found that the unaffected arm, which could be moved very well by itself, could not be moved so widely or forcibly when both arms were raised together. A somewhat similar condition has since been described by Grasset-Gausel (*R. n.*, 1905 and 1907) as regards the leg of the hemiplegic side. See also Bychowski, *N. C.*, 1907.

Monoplegia is seldom observed in lesions of the internal capsule, but an isolated facial paralysis (Diday, Duplay), and a crural monoplegia of this origin have been described. The latter is usually associated with sensory disturbances.

Lesions in the anterior limb of the internal capsule only produce symptoms of paralysis when they are adjacent to the knee. It has been already noted, however, that Brissaud localised the tract for emotional movements in this region. Vasomotor fibre bundles have also been assumed to lie in this zone.

Diseases of the *central ganglia* apparently give rise to paralytic symptoms only when they involve the internal capsule directly or by pressure, but this question, as already mentioned, is not yet definitely decided. Small foci limited to the corpus striatum or the optic thalamus may give rise to no symptoms of any kind. Disturbances of the mimic movements have been observed in a few cases of disease in the region of the *optic thalamus* (Oppenheim, Fraenkel, Miura, Raimann, Roulin, etc.). According to Nothnagel and Bechterew (see p. 651) conservation of the mimic movements of one side of the face is due to integrity of the optic thalamus on the other side. Small foci may cause symptoms of irritation (forced laughter), and larger ones loss of mimic expression, with conservation of the voluntary movements. On the other hand there are inhibitory tracts for the movements of mimic expression, which may be interrupted by focal diseases in the region of the central ganglia of both sides, so that laughing and weeping assume a spasmodic character (see chapter on pseudo-bulbar paralysis).

Consult also Oppenheim-Siemerling, *Charité-Annalen*, xii.; Sternberg (*Z. f. k. M.*, Bd. lii.); the bibliography in Wiesner (*W. kl. R.*, 1906); Deroubaux (*Journ. de Neurol.*, 1906), etc. The symptomatology of diseases of the thalamus will be further discussed in the following section.

We must further refer to certain *symptoms of motor irritation* most frequently observed in diseases of the optic thalamus, but which probably occur also in lesions of other parts of the brain. They are usually closely connected with hemiplegia, as they may follow, though they rarely precede it; they may, however, develop independently of it. These symptoms are *hemichorea*, *hemiathetosis*, and allied motor disorders.

[1] *Roum. méd.*, 1899, and *R. n.*, 1902. See also Loeper-Crouzon, *Nouv. Icon.*, xvii.

Hemichorea gives rise to involuntary movements in the limbs of one side of the body; these pass rapidly from one group of muscles to another, and produce a kicking or flinging movement in the limbs. They are aggravated by emotion and as a rule also by any attempt at voluntary movements. These have rarely the opposite effect. The form in which we are specially interested is known as *post-hemiplegic chorea*, as it follows upon hemiplegia; a pre-hemiplegic chorea is much less common. It may develop, for instance, in slow hæmorrhage into the optic thalamus (Charcot). It is also frequently associated with hemi-anæsthesia. Post-hemiplegic chorea may be confined to the arm or the leg (Mohr, Bernhardt, Oppenheim). Kussmaul has described under the name of *hemiballismus* a form or variety of hemichorea in which the involuntary movements of the arm have a more rhythmical character and suggest throwing movements.

Athetosis (Hammond) or hemiathetosis is also characterised by involuntary movements, which are most marked in the fingers and toes, whilst chorea affects the whole extremity. These are slow movements of separation and adduction, flexion and extension of the fingers (Fig. 298), which are usually continuous and persist even during sleep, sometimes with interruptions, or which occur only on the attempt at active movements, or, as I have sometimes seen, only during mental excitement. The fingers are not all moved at the same time or in the same direction, but show a very curious play of movement. They may be over-extended and spread out, whilst the hand is brought into a position of extreme flexion. Some of the fingers may be extended, whilst others are flexed, etc. These movements have been compared to those of the tentacles of polypi, although they are by no means so slow.

There is also a tonic contraction in the other muscles of the extremity. The arm may thus be pressed closely to the thorax, or may be brought into a position of excessive rotation. These positions are not persistently maintained but undergo a certain modification, which becomes specially apparent under the influence of emotion and voluntary movements (spasmus mobilis). Thus in walking the arm may be extended backwards or forwards; in one case, I saw it spasmodically raised above the head. There may also be slight spasmodic movements in the muscles of the face and tongue. In one of my cases the platysma was the only one involved, but was so to a high degree. Hypertrophy of the muscles has occasionally been observed in the extremity affected by the athetosis (Audry, Lannois, Bourneville, Brissaud-Hallion, Sicard, *R. n.*, 1905). The leg is usually less affected; there is as a rule plantar flexion and adduction of the foot, the great toe being often hyper-extended. Athetosis is distinguished from hemichorea by this kind of prolonged, though changing contraction of the muscles. We must, however, remember that there are motor affections which form a transition between chorea and athetosis, and which do not exactly correspond either to the type of hemichorea or to that of athetosis. These disorders may co-exist in the same case.

Further, tonic muscular contraction, constantly varying in intensity—spasmus mobilis—may alone be present, unaccompanied by choreic-athetotic movements, or it may be the chief symptom. This well-known affection, which I had described in the chapter on infantile cerebral paralysis in the second edition of this textbook, has been again described by Bechterew (*N. C.*, 1900) under the name of "hemitonia." On the other hand, I have seen in a child suffering from left hemiplegia with contracture, the sudden onset, whenever he became excited, of a movement of extension of the left arm and leg, rotation of the head and eyes towards the left, contraction of the left angle of the mouth, and deviation of the tongue towards the left—a spasmodic condition which only lasted for some moments, and which was therefore a kind of emotional hemitonia.

Except in some rare cases, the athetosis follows the hemiplegia, and it is specially apt to be associated with infantile hemiplegia (see

chapter on infantile cerebral paralysis). It follows the hemiplegia only after some months or even years, and only when a certain amount of

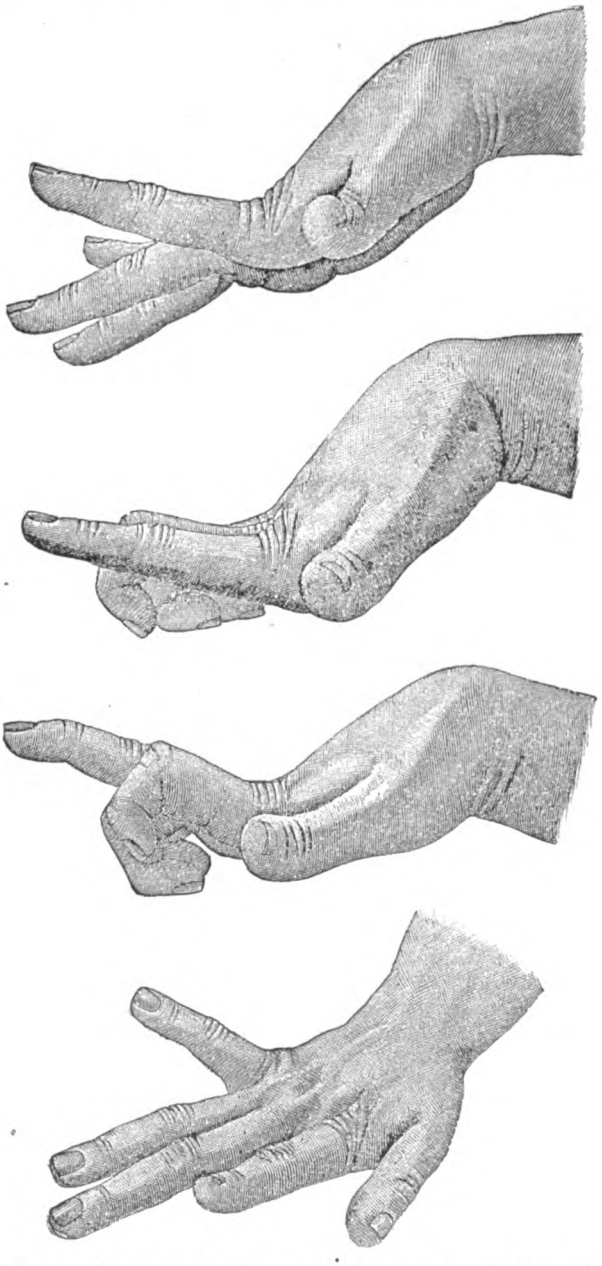

FIG. 298.—Position of the fingers in athetosis. (After Strümpell.)

active movement has been regained. These post-hemiplegic motor disorders usually develop simultaneously in the whole of one side of the

body, but a dissociated onset and distribution has been observed, especially in cases of neoplasms (Bonhöffer,[1] Sorgo [2]).

Hemichorea and hemiathetosis have been so frequently observed in localised lesions of the thalamus that there has been a tendency to attribute them to affections of this ganglion (Gowers, Stephan, Nothnagel). But on the other hand many cases seem to show that the symptoms are not caused by lesion of the optic thalamus itself, but by irritation of the neighbouring pyramidal tract. It has been suggested, therefore, that affections of the motor centres and lesions developing anywhere in the vicinity of the motor tract, in the pons, medulla oblongata, or elsewhere, might give rise to this disorder (Kahler and Pick,[3] with whom Greidenberg, Kolisch, Peritzeanu, and others agree). Charcot and Raymond think that a special bundle in the internal capsule has been implicated by the lesion.

The theory of Kahler and Pick and its modifications have not been supported by recent observations. They think that the lesions to which the hemichorea and hemiathetosis are due are localised in the central ganglia (middle and posterior area of the optic thalamus, the lenticular nucleus), the subthalamic region, the red nucleus, the superior cerebellar peduncles, and the cerebellum. Various theories have been constructed upon these facts as to localisation. Monakow suggests a continuous irritation passing from the optic thalamus and the mid-brain to the centripetal fibre bundles which pass to the cortex. Anton,[4] with whom Hartmann agrees, thinks that there is an antagonism between the optic thalamus and the lenticular nucleus, and regards the latter as an apparatus for inhibition of the automatic movements excited by the former. Lesions of the lenticular nucleus, or of the inhibitory fibres arising from it would therefore remove all restraint upon involuntary movements. Bonhöffer emphatically maintains that the symptoms of hemichorea and hemiathetosis are caused by focal lesions in the tract of the superior cerebellar peduncle, *i.e.* in the fibres which pass from the cerebellum through the superior cerebellar peduncle and the red nucleus to the thalamus, and their terminations. This view is supported by numerous observations, such as those of Leube, Pineles, Sander, Touche, Ramey, Adler,[7] Berger,[5] Hartmann, Homburger, D'Astros, Aufschlager,[6] Muratow,[7] and especially those of Halban and Infeld.[8] Bonhöffer has suggested various ways in which lesions so situated may produce post-hemiplegic motor disorders, but the view most generally accepted is that there is a modification of the current of stimuli which normally flow from the cerebellum through the tegmentum to the cortex. More recent writers (Sander, Pineles, Muratow, Haenel,[9] and others) have modified this hypothesis in various ways, and have in particular discussed whether the symptoms are irritative or paretic in character. Halban and Infeld, who, from their own observations and a careful review of the literature have practically confirmed Bonhöffer's teaching, have advanced a theory which endeavours to combine the fundamental ideas of Anton and Bonhöffer. Gordon Holmes [10] also agrees with Bonhöffer.

From all this it seems to me that we may construct the following

[1] *M. f. P.*, iii. and x.
[2] *N. C.*, 1902.
[3] *Viertelj. f. prakt. Heilk.*, 1879.
[4] *Jahrb. f. P.*, xiv.
[5] *Jahrb. f. P.*, 1903.
[6] *Z. f. k. M.*, Bd. li.
[7] *Rev. russ. de méd.*, 1903.
[8] *Obersteiner*, ix. See the bibliography here.
[9] *Z. f. N.*, xxi.
[10] *Br.*, 1904.

statement: Post-hemiplegic motor disorders, hemichorea, and hemiathetosis are caused by direct or reflex stimulation of the automatic motor centres in the optic thalamus, therefore (1) by small, irritative foci at certain sites in the thalamus; (2) by foci which are situated in the sensory fibre bundles passing into these thalamic centres, and particularly in the cerebello-thalamic tract or the tract of the superior cerebellar peduncle; (3) especially in childhood, by morbid foci which permanently cut off the motor cortical centres and thus cause the thalamic centres to acquire an excessive independence as regards involuntary movements, which is revealed by the chorea and athetosis (especially in infantile cerebral paralysis).

These statements make it clear that focal lesions in several situations can produce the phenomena in question, and also that motor disorders of different character may develop—depending perhaps upon the special site, the nature of the process, and the individual disposition.

Marburg (*W. kl. W.*, 1905) suggests that the fibres belonging to the system of the superior peduncles of the cerebellum have different functions, the tegmento-cerebellar being connected with athetosis, the thalamic with chorea, and the cortical with tremor; he is not himself, however, sure of this theory. He has come to the further conclusion that lesion of the rubro-spinal region usually causes bilateral symptoms of irritation of this kind, as the cases of Leube and Marina prove.

This view also makes it intelligible why symptoms of hemichorea and hemiathetosis are not necessarily accompanied by those characteristic of lesion of the pyramidal tracts (hypertonia, Babinski's sign, etc.), and why they are generally absent in pure cases (Bonhöffer, Halban-Infeld, Homburger, Oppenheim). Indeed there has frequently been diminution of the tendon reflexes, and various hypothetical opinions have thus arisen as to the relation of the tegmental tracts to the tendon reflexes and to Babinski's sign. Finally, it supplies a satisfactory explanation of the cortical origin of hemichorea observed in childhood and occasionally in adults.

Choreic contractions are said to have been experimentally produced by stimulation of the motor cortex with chemical poisons (creatinin).

I have had the opportunity of observing the onset of a hemiathetosis associated with hemianæsthesia and hemiataxia in a case of spinal disease. The case is not yet concluded, and further evidence is required before definite conclusions can be drawn from it. Kutner-Kramer (*A. f. P.*, Bd. xlii.) report a similar case. Siefert describes a "functional hemiathetosis" cured by psychotherapy (*A. f. P.*, xxxviii.). It is doubtful whether a case reported by Bauer as hemiathetosis, occurring in a woman during her confinement, and disappearing in two days, belongs to this category.

Idiopathic or primary athetosis[1] (athétose double) is a disease usually of bilateral symmetrical distribution, apparently of spontaneous onset, and showing no relation to hemiplegia, which occurs both in children and in adults. Trauma, chill, and agitation have been assigned as the causes in a few cases. I have twice seen the disease in members of one family, and once in a mother and daughter. The symptoms of athetosis

[1] We owe this description to Shaw (*St Barth. Hosp. Rep.*, 1873), Oulmont ("Étude clinique de l'athétose," *Thèse de Paris*, 1878), Osler (*Med. News*, 1888, and "On Chorea and Choreiform Affections," London, 1894), Audry ("L'athétose double," etc., Paris, 1892), Massalongo ("Sul l'athetos doppia," *Gaz. degli Osp.*, 1894), and Freud (Nothnagel's "Spez. Path.," ix.). See also Brissaud-Hallion (*R. n.*, 1893).

may be the only ones present, or they may be accompanied by idiocy, epilepsy, and other affections.

The movements more or less correspond to those of athetosis. Generally there is a kind of grimacing of the whole body, including the face, and there is an unusually strong and widespread tendency to associated movements, as I have already shown (*B. k. W.*, 1884), and as has been again recently pointed out by Lewandowsky (*Z. f. N.*, Bd. xxix.).

The disease may progress, may become arrested, or may even recover (in one case apparently owing to mercurial treatment). Its nature and cause are not yet clear; in the few cases examined post-mortem there were no pathological changes, apart from some irregularities in the convolutions (Dejerine-Sollier, Oppenheim). It is not even certain whether we are dealing here with an affection *sui generis*, or with a disease which belongs to the category of infantile cerebral diplegia (*q.v.*); in any case it appears to be closely allied to it, as Oulmont, Gowers, Audry, and Freud have specially pointed out. See also Goulard, *Thèse de Paris*, 1903.

Klempner (*N. C.*, 1906) describes a "forme fruste" of this disease, in which he succeeded in eliciting the "feed reflex."

Unilateral tremor and paralysis agitans posthemiplegia are less common forms of the condition of post-hemiplegic motor irritation.

Simple tremor has been described by Bristowe, Hoppe, and tremor of the type of paralysis agitans by Benedikt, Eisenlohr, Touche, Sorgo, B. Charcot, etc. I have seen typical paralysis agitans localised in the leg in a tumour which mainly affected the region of the lenticular nucleus, but the effect of the pressure may have extended to the region of the cerebral peduncle and tegmentum. Gordon Holmes (*Br.*, 1904) has written an exhaustive work on this affection; he ascribes it to the lesion of the cerebello-rubro-spinal system, and indeed regards the tremor as a symptom of cortical irritation which has arisen from the lesion of the tract in question. The statement of Gilles de la Tourette and Charcot that the tremor in this symptomatic form of paralysis agitans always increases under the influence of active movements, is not in accordance with my own experience.

A tremor resembling that of multiple sclerosis also sometimes occurs, as I myself have seen and as Ferrier, Bristowe, Mendel, Gowers, and especially Infeld and Sutherland have described. Babinski has observed a form of unilateral tremor in affections of the cerebellum or of the cerebellar peduncle. Simple rapidly successive contractions in certain muscle groups have also been observed, and a combination of tremor and ataxia have been found to follow the hemiplegia.

Associated Movements.—The paralysed limbs which are completely irresponsive to the control of the will are not infrequently set into action during voluntary movements of the muscles of the sound side or in such reflex movements as yawning, coughing, etc. Thus the paralysed hand may be clenched when the patient is shaking hands with the unaffected one, or a closed hand may open when he yawns. The reverse also happens, and active movements of muscles previously paralysed and still paretic, or the attempt to move the paralysed muscles is accompanied by movements of the same kind in the healthy side. These movements were termed compensatory by Senator.[1] Further, movements are sometimes observed in the paralysed upper limb when the patient tries to raise

[1] *B. k. W.*, 1892.

the leg of the same side, or *vice versâ*, some muscles of the lower extremity —the extensors of the foot and toes, for example—becoming contracted during articulation, movement of the arms, etc. The extensor hallucis is, in our experience, the muscle most frequently involved in these associated movements. In conditions of spastic paralysis, powerful flexion of the thigh is usually accompanied by tonic movement of the tibialis anticus (Strümpell's tibialis phenomenon), but this is by no means constant (see p. 177). These associated movements may be particularly marked in the forms of paralysis which occur in early childhood, and may be developed to such a degree that all the movements are bilaterally symmetrical (Westphal).[1]

Compensatory movements are mainly due to the fact that the effort to set the paralysed muscles in motion calls for an excess of innervating energy, and that the impulse is thus transmitted to the opposite side. The occurrence of associated movements in paralysed limbs is attributable mainly to the fact that even under normal conditions—particularly in childhood—each hemisphere is connected with the limbs of both sides. Monakow assumes that there is in addition exaggerated irritability of the deep motor centres separated from the cortex. A symptom which has struck me as being very frequent in diplegia, viz., *excessive timidity* or exaggerated auditory motor reaction of the patient, on account of which a slight noise evokes a reflex movement of abnormal vehemence and extent (violent shaking of the whole body), corroborates this view (*M. f. P.*, xiv.). The chapter on infantile pseudo-bulbar paralysis may be consulted with reference to the exaggeration of certain reflexes (feed-reflex, etc.).

Thomson (*Br.*, 1903) gives valuable details as to associated movements.

Some very remarkable cases of congenital associated movements (synkinesis) in otherwise normal persons are described by Thomayer, Damsch,[2] Levi, Fragstein,[3] Medea-Hanau,[4] Fuchs,[5] and Brissaud-Sicard.[6] A symptom is noted by Fragstein which is most difficult to interpret, viz., the bilateral character, not of active movements merely, but also of passive movements and of those provoked by electricity. This tendency to associated movements may descend from one generation to another. From my own experience I should be inclined to regard certain forms of associated movements as stigmata of degeneration. The question of these movements has been carefully studied by O. Förster [7] and H. Curschmann.[8]

The latter insists that the tendency to associated movements is a physiological sign, which is very marked in childhood and disappears gradually but not entirely, and may specially persist in the ends of the extremities, the fingers and toes, in the movements of separation and adduction.

With regard to so-called diadokokinesis, see the section on cerebellar diseases.

. . . . . .

If the motor tracts are affected at lower levels a hemiplegia is produced which, in so far as the paralysis of the extremities is concerned, is similar to that due to a capsular lesion, whilst the cranial nerves may show a different condition. In lesions of the *crus cerebri* the otherwise *typical hemiplegia* is associated as a rule with paralysis of the *crossed oculomotor* nerve ; that is to say, lesion of the ventral portion of the left crus

[1] *A. f. P.*, iv. [2] *Z. f. k. M.*, 1891, Supplement. [3] *M. f. P.*, x.
[4] *Rev. de Psychiat.*, 1902. [5] *W. kl. R.*, 1905. [6] *R. n.*, 1905.
[7] "Die Mitbewegungen," Jena, 1903. See also Oppenheim, *B. k. W.*, 1884 ; Camus, *Thèse de Bordeaux*, 1885 ; F. Sander, *Dissert.*, Halle, 1894.
[8] *Z. f. N.*, xxxi.

cerebri gives rise to paralysis of the *left oculo-motor nerve and to right-sided hemiplegia* (superior hemiplegia alternans or Weber-Gubler paralysis). This is explained by the anatomical relationships of the parts involved (Fig. 279). A tremor may also appear in the paralysed limbs, of the type usually of the tremor in paralysis agitans (Benedict's symptom), chiefly when the process affects the tegmental area of the cerebral peduncle. Cases of this kind have been reported by Gilles de la Tourette and J. B. Charcot,[1] by Sorgo, Raymond-Cestan,[2] Gordon Holmes,[3] and in particular by Halban-Infeld. We need not here discuss the other symptoms which may be caused by extension of the focus into the red nucleus, the lemniscus, etc. It should, however, be remembered that oculo-motor paralysis will be partial or complete according to the special localisation of the lesion, that should the focus extend beyond the middle line, some bundles of the oculo-motor of the opposite side will be involved, and that a very small number of cases of bilateral hemiplegia alternans superior, due to focal lesions of both crura cerebri, have been observed (Souques).

Marburg[4] has studied this question very thoroughly. He distinguished three groups of symptoms, which correspond to the three levels of the region of the anterior corpora quadrigemina, viz. :—1. Foci of the pes pedunculi—in its posterior-median segment—produce paralysis of the oculomotor nerve with hemiplegia of the opposite side, in which the seventh and twelfth nerves usually take part; 2. Foci in the tegmentum give rise to Benedict's symptom, namely, unilateral oculomotor paralysis with tremor, chorea, or athetosis of the opposite side. Hemi-ataxia may also develop. 3. Foci in the corpora quadrigemina cause oculomotor paralysis of one or both sides, with ataxia, chiefly of the cerebellar type (Nothnagel). The question whether auditory affections occur in cases of foci limited to the region of the corpora quadrigemina, still requires further study, in spite of the work of Siebenmann (*Z. f. O.*, 1896) and Wienland (*A. f. P.*, xxvi.). These auditory symptoms are probably due to involvement of the lateral fillet or the internal geniculate body. The appearance of ambylopia, etc., under these conditions also requires further explanation.

As the oculomotor nerve passes through the root bundles of the cerebral peduncle, it will be seen that the paralysis will sometimes be total (Gubler, Luton, Léteinturier, Alexander) and sometimes partial (Kahler-Pick, Oyon, D'Astros; bibliography in Marburg). Although in foci limited to the pes pedunculi the affection of the oculomotor nerve is almost always unilateral, it is often bilateral when the foci occupy the tegmentum or the corpora quadrigemina. In the latter case the symptom of paralysis of the upward and downward conjugate movements of the eyes is often observed (see further on).

The fact that hemianopsia on the opposite side may appear along with oculomotor paralysis, especially in tumours (Wernicke, Mahaim, Blessig, Raymond, *Gaz. des hôp.*, 1902, Marie-Léri, *R. n.*, 1905) is due to the proximity of the external geniculate body and the optic tract to the oculomotor nerve and nucleus. Hemianopic immobility of the pupils has occasionally been observed in these cases (Wernicke). One such case has been thoroughly investigated post-mortem by Rossi-Roussy (*Nouv. Icon.*, xix.). Achard-Levi have described (ref. *N. C.*, 1902) one case of oculomotor paralysis without hemiplegia in softening of the crus cerebri.

Focal lesions in the *uppermost portion of the pons* give rise to simple hemiplegia, and the site of the lesion is only definitely indicated by involvement of other tracts which traverse the pons.

If the lesion is situated below the decussation of the central facial tract—in the neighbourhood or at the level of the nucleus—the paralysis will affect the facial nerve on the same side as the lesion and the extremities on the opposite side. This constitutes the *hemiplegia alternans* (Millard-

[1] *Semaine méd.*, 1900.
[2] *R. n.*, 1902. See also Lévi-Bonniot (*R. n.*, 1905).
[3] *Br.*, 1904.
[4] *W. kl. W.*, 1905.

Gubler[1] type) which is so characteristic. In a similar way, though extremely seldom, hemiplegia alternans involving the hypoglossal of one side and the extremities of the other may appear; the tongue then deviates towards the unaffected side. There is also a hemiplegia alternans in which the abducens or the associated ocular muscles on the affected side are involved with the extremities and eventually with the facial of the other side (type of Foville). (Raymond[2] has lately drawn fresh attention to this form). Unilateral affection of the auditory nerve and the motor trigeminus may be combined with alternating paralysis of the extremities. Neuroparalytic keratitis may also develop in alternating paralysis of the sensory trigeminus, as shown by the cases of Gubler, Jackson, Bernhardt, Oppenheim, and Marie-Crouzon. This whole question is discussed in the section on diseases of the pons.

A symptom which sometimes occurs in brain diseases is the so-called *conjugate deviation of the head and eyes* towards one side (Prevost,[3] Landouzy-Grasset). In diseases of the cerebrum which commence with paralysis of the opposite side of the body, the head and eyes are very often, especially at the onset of the acute process, deviated towards the opposite side, *i.e.* towards the side of the lesion. This symptom is due to paralysis or weakness of the muscles which rotate the head and eyes towards the side of the hemiplegia, therefore towards the side opposite to the lesion. Murri, on the other hand, ascribes it to an irritative condition of the opposite cerebellar hemisphere.

In spasmodic conditions, again, the head and eyes deviate towards the side of the spasm.

This symptom is particularly common in focal diseases of the frontal lobe, specially of the hinder end of the second (and first) frontal convolution, but there is no doubt that it may also originate from other cortical regions (angular gyrus, possibly the occipital lobe), and it must therefore be used as a localising sign only with great reserve. According to E. Müller (*Z. f. N.*, xxii.) it may be ascribed to the frontal lobe when it is particularly marked, when it recurs in repeated attacks, and is always directed towards the same side, etc.

The attempt made by French writers, such as Bard (*Semaine méd.*, 1904), Dufour (*R. n.*, 1904), and Grasset (*Semaine méd.*, 1904) to prove that conjugate deviation occurring in affections of the cerebrum is related to and dependent upon hemianopsia, may be considered a failure, as Dejerine and Roussy have already shown (*R. n.*, 1905).

In exceptional cases the head is directed to one side and the eyes to the other (Prévost, Roussy-Gauckler). With regard to the question of paralysis of conjugate deviation of the eyes, see also Brissaud-Péchin, "Hémiplégie oculaire," *Prog. med.*, 1904; and Gaussel, *Rev. de Med.*, 1905.

In pontine disease the eyes often deviate towards the side opposite to that of the lesion. This symptom, which had been established by Foville,[4] was formerly explained by the assumption of a centre for the lateral movements of the eyes towards the same side in the abducens nucleus or its neighbourhood (Wernicke,[5] etc.). But the view that the abducens nucleus itself represented such a centre proved untenable, as it was found to be intact in various cases of pontine conjugate deviation (Quioc, Hunnius,[6] Garel,[7] Senator). Grasset and Gaussel (*R. n.*, 1906)

[1] *Gaz. hebd. de méd.*, 1856 and 1859.

[2] *R. n.*, 1895. Consult also Oppenheim, "Hirngeschwülste," 2. Aufl., Abschnitt Brücke; also Hirsch, *Z. f. Aug.*, 1903; H. Schlesinger, *Jahrb. f. P.*, xxii.; Varet, *Thèse de Paris*, 1905.

[3] "De la déviation conjugée.," etc., *Thèse de Paris*, 1868. See the bibliography on this subject in Uhthoff, Graefe-Sämisch, "Handbuch," 2nd ed., T. ii. Bd. xi., p. 600.

[4] *Gaz. hebd.*, 1859.

[5] *B. k. W.*, 1876; *A. f. P.*, vii.

[6] "Zur Symptomat. der Brückenerkr.," etc., Bonn, 1881.

[7] *Rev. de Méd.*, 1882.

still, however, maintain this view. Hunnius, Bleuler,[1] Jolly,[2] and others attribute this paralysis to lesion of the posterior longitudinal bundle, on the assumption that a tract passes down through it to the nucleus of the abducens of the same side, where it turns, forms a loop, and again ascends to the oculo-motor nucleus. This fasciculus may either terminate in the oculomotor nucleus of the opposite side, or in the cell groups of the same side which send their roots into the crossed internus. Anatomical investigations and clinical cases by Duval, Bechterew, Gee-Tooth,[3] Kohnstamm, Henneberg,[4] Raymond,[5] Seggels, Bruce,[6] and others may be interpreted in this sense (see p. 656). Spitzer,[7] Jolly, Monakow, and others certainly adhere to the view of a centre for conjugate deviation of the eyes which they localise in the mid-brain, or with Adamük in the region of the corpora quadrigemina (according to Monakow it is represented not by circumscribed ganglion groups, but by scattered ganglion cells which act as intercalary cells)—but they attribute the associated conjugate paralysis in diseases of the pons to lesion of the *posterior longitudinal bundle.* The majority of recent observations are in favour of this view, according to which pontine paralysis of conjugate deviation should be regarded as of supra-nuclear origin.

We may therefore take this fact as established. It only remains to decide on the one hand whether the tracts coming from the cortical centres must first enter a special centre for conjugate deviation situated in the region of the corpora quadrigemina, or whether they pass directly into the posterior longitudinal bundle and thence to the oculo-motor nuclei. In any case this cortico-nuclear tract undergoes decussation (complete or partial) before it enters into the posterior longitudinal bundle.

As each cortical centre has a bilateral association with the centres for conjugate deviation or with the nuclei for the ocular muscles concerned in this movement, although this association is mainly with that of the opposite side, a permanent paralysis of conjugate deviation will hardly ever occur in diseases of the cerebrum, except as the result of a bilateral lesion (see section on pseudo-bulbar paralysis). Cases of this kind have been published by Wernicke,[8] Oppenheim,[9] Tiling, Ballet,[10] Roth,[11] and others. Under these conditions it is only *voluntary* lateral movement of the eyes that is impossible, whilst the automatic and reflex lateral movement, and in particular the movement determined by the auditory apparatus, remains unimpaired, as I was able to demonstrate in one case. Roth and Bielschowsky [12] in particular, have described the following method of determining supranuclear paralysis of conjugate deviation from that due to affection of the nuclei and roots. The patient is asked to gaze straight forward, and the head is then passively and slowly rotated towards the unaffected side; the eyes are thus brought into the extreme lateral position, which they cannot voluntarily assume. Bárany [13] who has confirmed this symptom, attributed it to stimulation of the semicircular canals, which reflexly control the position of the eyes.

Cantonnet-Taguet (*R. n.*, 1906) have dealt with this question without being aware of our contributions to it. They adhere firmly to the view of a centre for conjugate deviation in the

[1] *A. f. kl. M.*, Bd. xxxvii. [2] *A. f. P.*, xxvi. [3] *Br.*, 1898.
[4] *Charité-Annalen*, xxvii. [5] *Gaz. des hôp.*, 1903, and *R. n.*, 1904. [6] *R. of N.*, 1903.
[7] *Obersteiner*, vi., 1899. [8] *A. f. P.*, xx. [9] *Fortschr. d. Med.*, 1895.
[10] *R. n.*, 1906. [11] *N. C.*, 1901. [12] *D. m. W.*, 1903.
[13] *M. f. Ohr.*, 1906; *A. f. Ohr.*, 1906; *M. m. W.*, 1907; and, in particular, "Unters. über den vom Vestibularapparat. des Ohres refl. ausgel. Nystagmus," etc., Berlin, 1906.

mid-brain and think that according to the site of the lesion three forms of paralysis of these movements can be distinguished: 1. Loss of voluntary conjugate deviation with conservation of reflex and automatic movements; 2. Loss of the reflex and automatic conjugate deviation with conservation of voluntary control; 3. Paralysis of all the movements of conjugate deviation, voluntary, automatic, and reflex.

Much study has of late been devoted to the question of the influence of the labyrinth, or of the vestibular nerve on the movements of the eyeballs. The anatomical relations have already been described on pp. 645 and 663. Bárany in particular has investigated with much care the evidence of these connections and their diagnostic value. He agrees with Wanner and others. The nystagmus which follows repeated rotation on the vertical axis—in looking towards the opposite side (see below)—is due to stimulation of the semicircular canals, which is transmitted through the vestibulo-oculo-motor tracts to the muscles of conjugate deviation of the eyes. Destruction of the labyrinth suspends this influence. Labyrinthine movement of the eyes in the form of rhythmic nystagmus may be produced through the same tracts by syringing the ear with hot or cold water. This result does not follow if there is a lesion of the labyrinth or of this reflex tract. Galvanic vertigo and the nystagmus which accompanies it may also be produced in this way. As Bárany has shown, therefore, corresponding methods of investigation may be employed to determine whether a lesion is situated in the labyrintho-vestibular region or in the tracts which pass from the vestibular nerve through the posterior longitudinal bundle to the oculo-motor nuclei, or external to these. These questions certainly call for further investigation, but there can be no doubt that this theory is in the main correct. Paralytic symptoms of acustico-oculo-motor origin have also been described, *e.g.* by Steyskal and by M. Sachs (*W. kl. R.*, 1904), but it should not be forgotten that the labyrinth of the ear may be the origin of irritative symptoms in the form of nystagmus, and perhaps of tonic deviation of the eyes and of transient diplopia, but it is never the source of true paralysis of the muscles from the conjugate deviation of the eyes. True, persistent paralysis of these movements is, therefore, a certain sign of affection of the pons, which of course may arise from pathological processes in its neighbourhood (cerebellar tumour, etc.).

Paralysis limited to one internal rectus as a muscle of lateral movement, convergence being unimpaired, has been described by Bielschowsky and Fischer (*Prag. med. Woch.*, 1905), and attributed to lesion of the corresponding tract in the posterior longitudinal bundle.

As convergence is usually intact in paralysis of lateral conjugate deviation—the internus failing therefore only in lateral movements, but acting normally under other conditions, and also sometimes under monocular testing (Féréol, Graefe, Wolff)—it seemed necessary to assume the existence of a special centre and a special tract for the movement of convergence. Spitzer on the other hand has suggested that in such cases there is simply a paresis of the internus, which in the interest of clear vision is overcome by powerful innervation when convergence is attempted, whereas the attempt to overcome it during lateral movement of the eyes would only aggravate the diplopia.

Bilateral lesions of the pons, or unilateral foci which extend beyond the raphe and injure the longitudinal bundle on both sides may also give rise to paralysis of conjugate deviation in both directions.

Paralysis of the upward and downward movements of the eyes is much less common, although a number of such cases have been recorded by Nieden, Graefe, Schröder, Parinaud, Smith, Babinski, Thomsen, Sauvineau, Nogues-Sirol, Raymond, Hänel, Bruce (*Trans. Med. Chir. Soc.*, Ed., xix.), Kornilow (*Z. f. N.*, xxiii.), Lichtheim, Marburg, Gruner-Bertolotti (*Nouv. Icon.*, xviii.), etc. The paralysis usually affected the upward movement (I have found this as a congenital condition in one case), but we have very little definite knowledge as to its cause. There seems, indeed, to be a definite centre and a corresponding tract for these movements, and a lesion of this hypothetical centre or a bilateral involvement of the corresponding supranuclear tracts may possibly be the cause of this paralysis. Indeed this form of paralysis has been specially observed in affections of the region of the corpora quadrigemina in the neighbourhood of the aqueduct of Sylvius, although it is not one of the symptoms of pontine disease. Spiller has dealt exhaustively with this question (*Journ. Nerv. and Ment. Dis.*, 1905), and has traced the

paralysis to lesion of the corresponding supranuclear fibres. See also Tödter (*Kl. Mon. f. Aug.*, 1906).

We may here refer shortly to a peculiar form of ocular affection, previously described, I believe, by Magendie, and more recently by English writers, such as Stewart and Holmes, and Ballance ("Some Points in the Surgery of the Brain," Lond., 1907), namely, "skew deviation," in which the eye of the affected side is turned downwards and inwards and the other upwards and outwards (Fig. 299). This is chiefly observed after surgical operations on the cerebellum, when the eyes were directed towards the opposite side.

We have already in the general part referred briefly to *nystagmus*. This symptom occurs under many conditions, and in diseases of very different character and localisation. We have first of all a *physiological* nystagmus, which may occur in healthy individuals under various conditions: 1. In the fixation of rapidly moving objects, *e.g.* in the attempt to fix an object out of a moving railway carriage; 2. When tested as follows: If we turn a person six to ten times round on his

FIG. 299.—(After Stewart and Holmes.) Skew deviation of left eye.

vertical axis and then stop him, marked nystagmus will appear when his eyes are turned towards the opposite side, *e.g.* if he has been turning round to the left and then directs his eyes to the right. This nystagmus originates from the labyrinth, from the movement of the endolymph, and from the displacement of the cupulæ, etc., which it causes. Wanner,[1] has drawn special attention to the diagnostic value of the nystagmus thus produced. 3. We may also regard as physiological the nystagmus which is produced by galvanisation, by artificial increase of the pressure in the ear (blowing air into the external auditory meatus), or by syringing the ear with hot or cold water. Barány has pointed out the diagnostic importance of this test.

Among the forms of *pathological* nystagmus, we must mention first the congenital, that associated with congenital amblyopia, albinism, etc. It has usually a rotatory, oscillating character, is present during rest, but is increased during fixation.

[1] "Über die Erscheinungen von Nystagmus bei Normalhörenden, Labyrinthlosen und Taubstummen," München, 1901.

Nystagmus may accompany acquired amaurosis (Rählmann), but is then merely an accessory symptom.

Working in a bad light and with the eyes in an abnormal position seems to be the cause of *miners' nystagmus.*

See the latest contribution to this subject by Reid, *Br.*, 1906.

Nystagmus may also be caused by disorders of the circulation in the cranial cavity, such as a suddenly developing anæmia or hyperæmia.

It may also be found in some conditions of intoxication, such as acute alcoholism carbolic acid or cresol poisoning (Weyl).

Nystagmus is also of very great importance as a *focal symptom* in lesions of certain parts of the brain. It is due as a rule to affections of the cerebellum, the cerebellar peduncle, the nuclei of the oblongata which are related to the vestibular nerve, the posterior longitudinal bundle, and the *vestibular nerve* itself. If we note, in addition, that affections of the terminal ramifications of the vestibular nerve in the labyrinth, or of the *labyrinth* itself tend to produce nystagmus, we have before us the main conditions for the production of this symptom.

The anatomo-physiological relations of the labyrinth or the vestibular nerve to the nuclear area at the lateral angle of the fourth ventricle already described, and through it to the cerebellum of one side and to the nuclei of the ocular nerves of the other, suggest that this area has a definite physiological function, and that nystagmus is the indication of its irritation or lesion. Various attempts, some of which have already been referred to, have been made to discover the *modus operandi* of this mechanism, and to make it available for diagnostic purposes.

The question has been studied from this point of view by such aurists as Wanner, Herzfeld, Jansen, Hinsberg,[1] Krotoschiner,[2] Passow,[3] Stein,[4] and notably by Barány.[5] They have endeavoured to show that destruction of the labyrinth is revealed by absence of physiological and artificial nystagmus (see above) and by certain forms of disorder of the equilibrium. Their explanations of these connections are, however, by no means convincing. The advance in our methods of investigation, to which neurologists have also contributed, undoubtedly tends to further progress in the matter of diagnosis.

In attempting to establish a diagnosis of disease of the posterior cranial fossa, therefore, it is advisable not to be content merely with ascertaining the mere existence of cerebellar ataxia and nystagmus in the lateral position of the eyes, but to test also the condition of equilibration in active and passive rotation round the axis of the body, in various positions of the head, in standing on one foot (a test which I have used for many years), in standing on an inclined plane, in hopping backwards, etc. There is hardly any need for special apparatus, such as the swinging chair and the goniometer.

Whether and in how far it is possible to distinguish by more refined investigations of this kind between diseases of the labyrinth and those of the vestibular nerve, and between affections of this nerve and those of the cerebellum, etc., the future alone will show.

[1] "Labyrintheiterungen." Habilitationsschrift, Wien, 1901.
[2] *Z. f. Ohr.*, Bd. li. [3] *B. k. W.*, 1905. [4] *Z. f. Ohr.*, 1905.
[5] "Unters. über den vom Vestibularapparat des Ohres reflektorisch ausgelösten Nystagmus und seine Begleiterscheinungen," Berlin, 1906; also "Physiologie und Pathologie des Bogengangapparates beim Menschen. Klin. Studien," Leipzig-Wien, 1907.

Recent investigations have proved that galvanic reaction of the labyrinth or the vestibular nerve may possibly be of diagnostic value. Hitzig had previously noted that a transverse passage of the galvanic current through the skull with a current of about 1-1·5 MA. was followed by a reaction in which the head of the person under examination was inclined towards the side of the anode. Babinski (*Compt. rend. de la Soc. de Biol.*, 1901) then suggested that in unilateral diseases of the ear the head was inclined towards the *affected* side, whether it was connected with the anode or with the cathode. Mann (*Med. Klinik*, 1907), from his own investigations, modified this teaching by pointing out that this reaction of Babinski only followed if the internal ear or the vestibular apparatus were affected.

Cassirer and Loeser (*N. C.*, 1908) found that pathological nystagmus produced by rotation round the axis of the body in one direction (right), whilst the eyes are kept turned to the opposite side (left), may be absent. They explain this symptom by assuming that the pathological stimulus which gave rise to the primary nystagmus in the vestibular apparatus of one side, is more than compensated by the adequate stimulation of the other.

The occurrence of *nystagmus in diseases of the spinal cord* is without doubt an indication of the involvement of the cerebro-spinal nervous system, or the cerebellum, or the parts adjoining it (in disseminated sclerosis, Friedreich's disease, etc.).

Disturbances of the Sensibility

As we have already shown on pp. 626 *et seq.*, the cortical sensory centres are situated mainly or entirely in the posterior central convolutions—and probably in portions of the parietal lobe. Affections of the Rolandic area, therefore, give rise in many cases not only to paralytic symptoms, but also to modifications of sensibility. These very rarely amount to complete hemianæsthesia. There is usually paræsthesia, and also slight blunting of sensation, chiefly at the distal parts of the extremities, which may be so slight that careful examination is needed to detect it. It may involve the tactile sense alone, or may extend to several or all varieties of cutaneous sensibility. Monakow thinks the sense of position is most constantly affected, and Bonhöffer is of the same opinion. I have seen peroneal paralysis follow a surgical incision and puncture in the area of the leg centre, in which the power of localisation was only affected in the foot, sensibility being otherwise intact. Complete and persistent analgesia and thermanæsthesia can apparently never be caused by circumscribed cortical foci. Cortical anæsthesia may affect mainly the sense of position. When the sense of position is affected by itself it appears that the lesion is generally in the parietal lobe (see p. 628). This symptom may then be associated with hemiataxia.

F. Krause (*D. Klinik*, etc., 1904) has seen the bathyanæsthesia persist longest after operation upon the central region. A communication by Fischer (*M. f. P.*, xviii.) may also be considered in this respect.

Cortical hemianæsthesia is most marked in large hæmorrhages in the meninges, which compress the greater part or the whole of the cortex of the centro-parietal region. This does not exclude the possibility that the pressure may affect the deep, subcortical parts. In one of my patients who had a tumour of the parietal lobe, the hemianæsthesia was so complete that he did not feel but only saw the spasms which affected one side of his body (*Mitt. a. d. Grenzgeb.*, vi.).

Considerable areas of the motor region may undoubtedly be enucleated without causing any permanent impairment of the sensibility, as cases of Charcot, Pitres, Wernicke, Oppenheim, Monakow, Marie and others show. There is therefore a cortical hemiplegia without hemianæsthesia, and on the other hand there may be cortical hemianæsthesia without marked hemi-

plegia (Monakow, Prevost, Oppenheim, Henschen, F, Müller,[1] Lejonne-Egger); but it is practically always associated with hemiataxia (Oppenheim).

In complete destruction of the centro-parietal region, all forms of sensibility may be abolished, but this according to Monakow is in part due to diaschisis (see p. 623), and as a rule recovery is complete. F. Müller has made a careful study of the manner and order in which the symptoms improve. He thinks that restitution is partly due to compensatory action of the corresponding centres of the opposite hemisphere.

We have no definite knowledge of sensory disorders occurring in diseases of other regions of the cortex.

A peculiar sensory disorder has been noted by Wernicke,[2] in lesions of the motor zone or of the posterior central convolution, and by Oppenheim (Mills,[3] Burr, etc.) in lesions of the parietal lobe. Although the sensibility of the hand is practically intact, objects cannot be recognised by palpation (tactile paralysis, mind-anæsthesia). There seems to be a loss of power to deal with the memory pictures acquired by means of palpation.

I have sometimes found this symptom present at the first examination, and absent shortly after—when I tried to demonstrate it at a clinical lecture. It is therefore apparently due merely to impairment of the capacity to direct the attention to the affected side. I have found, moreover, that this symptom is very frequent in the hemiplegia of early childhood and especially in that dating from birth, and that it is due in these cases to the fact that no tactile memory pictures have been acquired by means of the paralysed hand. This factor has been pointed out by other writers (Claparède, Dejerine,[4] Burr), who were unaware of my communications on the subject.

Flechsig and Monakow endeavoured to explain this symptom as a loss of the co-ordination of the elementary sensations of the skin, muscles, joints, etc., which lead to the formation of correct stereognostic perceptions. That is to say there is a disturbance of sensory associations. This is also assumed by Bonhöffer,[5] who localised the lesion, in its pure form at least, in the anterior central convolution. Kutner[6] agrees with him. Kramer[7] takes a similar view, but agrees with Wernicke in localising the affection in the middle (third of the anterior) central convolution. Hartmann agrees with Storch as to the importance of the ideas of direction and of the impairment of the sense of space in regard to this perceptive process.

It is apparent from these facts that Wernicke's sensory affection cannot be sharply distinguished from the so-called "astereognosis" or stereoagnosis, the loss of the power to recognise the form and shape of bodies by palpation (see p. 48). In most cases this is, of course, merely a direct consequence of the elementary disturbances of sensibility. Tactile anæsthesia, and still more bathyanæsthesia, are specially apt to cause impairment of the stereognostic function. This function does

[1] *Volkm. Samml. kl. Vortr.*, 1905.

[2] *Arb. a. d. Bresl. psych. Klinik*, 1895.

[3] For references to the literature see p. 627. Contributions to the subject have also been made by Knapp (*M. f. P.*, xiv.), Bonhoffer (*Z. f. N.*, xxvi.), Kutner (*M. f. P.*, xvii. and xxi.), Schittenhelm (*A. f. kl. M.*, Bd. lxxxv.). Kutner describes one pure case in which the symptom was present along with absolutely unaffected sensibility; he interpreted it as an associated, transcortical affection, and regarded it as the result of slight superficial changes in the cortex of the tactile zone of the hand.

[4] "Sémiol. du Systeme nerv.," 1900.

[5] *Z. f. N.*, xxvi.

[6] *M. f. P.*, xvii. and xxi.

[7] *M. f. P.*, xix.

not, however, represent a simple sensory process, but rather a combined psychophysical act, in which, in addition to the elementary perceptions, a part is played by associative factors, memory, and especially by the reproduction of optical memory pictures. There must therefore be cases in which stereognostic perception is impaired in spite of the sensibility being intact, because the power to associate simple sensations and to elaborate them into ideas has been destroyed. We may take it that focal lesions which cut off the sensory sphere from the other regions of the cortex, especially from the occipital lobe—*e.g.* deep focal diseases of the parietal lobe, such as tumours—are particularly apt to give rise to this symptom. This seems to be proved by the cases published. The subject has been specially studied of late by Redlich,[1] Monakow, Oppenheim, Bruns, Gasne, Sailer, Dejerine, Long, Williamson, Soury, Verger,[2] Burr,[3] Mills, Heveroch,[4] Chipault, Chrétien,[5] Teller-Dercum,[6] Bullard,[7] Mills, and Weisenburg.[8] Some of these writers, and also Markova,[9] Walton and Paul,[10] and Diller,[11] localise the site of "astereognosis" in the central region, especially in the posterior central convolution, and in this they agree with Wernicke (and his pupils). Nevertheless it is still doubtful whether there are simple tactile memory pictures so independent that they can be called up without the co-operation of areas other than the motor zone or the tactile memory sphere. I cannot very well imagine this. Even were this the case, it would still be difficult to understand how affections of the corresponding cortical area should efface these memory pictures alone, without impairing the elementary perceptions. It seems to me therefore that Wernicke's tactile paralysis is practically identical with the astereognosis just described. In any case I do not know how one could be diagnosed from the other. We have therefore a *pure astereognosis* identical with Wernicke's tactile paralysis, in which the loss of the power of tactile recognition is not caused by impairment of the elementary perceptions, and another *secondary* form, which is simply the result of anæsthesia. Whilst the latter may naturally be due to affections of the sensory centres and nerve tracts, the former is probably the result of lesion of the fibre bundles or association tracts which connect the sensory cortical sphere with other cortical territories, especially the optic region. It is conceivable that this interruption may take place both in the immediate neighbourhood of the motor-sensory centre of the hand—*i.e.* in the area of the posterior central convolution or directly behind it (Wernicke)—or in the parietal lobe (Oppenheim, Bruns, Mills, etc.). Others think that the tactile memory pictures are associated with a special cortical field (central convolutions ?), lesion of which may produce a pure astereognosis; this then is often combined with motor and generally with slight sensory disturbances (monoplegia, Jacksonian epilepsy, impairment of localisation and the sense of position, etc.). Wernicke, Verger, Bonhöffer, Kramer, etc., are in favour of this origin and especially of localisation in the central area. Although from my own experience I must still regard the parietal lobe as being the site, yet I have in mind not a centre for tactile memory pictures, but rather an interruption of

[1] *W. kl. W.*, 1893.
[2] *R. n.*, 1902.
[3] *Journ. Nerv. and Ment. Dis.*, 1898.
[4] Ref. *N. C.*, 1902.
[5] *Thèse de Paris*, 1903.
[6] *Journ. Nerv. and Ment. Dis.*, 1901.
[7] *Journ. Nerv. and Ment. Dis.*, 1904.
[8] *Journ. Nerv. and Ment. Dis.*, 1906.
[9] "La perception stéréognostique," *Thèse de Genève*, 1900.
[10] *Journ. Nerv. and Ment. Dis.*, 1901; *Br.*, 1901.
[11] *Br.*, 1902.

the association tracts which pass through this area and which results in impairment of the sense of position and is of essential importance as regards tactile recognition. Moreover, recognition of form and shape may be impaired, to a certain degree at least, by affections of the motor power (Long, Markova).

Recent French literature shows an extraordinary confusion of ideas as regards this question, tactile paralysis and astereognosis being confounded with tactile aphasia. See Egger, *R. n.*, 1907, and Dejerine's criticism, *R. n.*, 1907.

Sensory symptoms of much greater severity are caused by lesions of the *sensory paths*. Thus extensive softenings in the subcortical white substance of the central and parietal region may more or less completely destroy the sensibility of the opposite side. As a rule the analgesia is neither total nor persistent. Morbid foci which destroy the posterior zone of the internal capsule or the corresponding ganglion masses in the optic thalamus (see p. 642), or the thalamo-cortical fibre bundles, cause *hemianæsthesia* of the opposite side of the body. When this is complete, it extends over all the skin and mucous membrane of one side and is limited by the middle line. I cannot confirm the opinion that the cornea is always spared (Grasset). The anæsthesia is not usually equally distributed over all parts ; it is marked at certain sites, and is only detected at others by repeated comparative examinations. According to Dejerine, Verger, Ferenczi,[1] and my own observations, it increases as a rule in intensity from the proximal towards the distal parts of the extremities, but Schaffer[2] has shown that its distribution may not be of this type. It may involve some or all the qualities of sensation. The sense of position may be entirely abolished. It was found by Verger and by Dejerine and his pupils to be as a rule very greatly reduced, but this has not been my experience. The analgesia and thermanæsthesia are never absolute, but on the other hand the power of differentiating the corresponding stimuli is often diminished and sometimes abolished. In a few cases I have found the senses of pain and temperature alone affected, whilst, inversely, in one case all forms of sensation were impaired, except that of heat, which was marked and even exaggerated. Isolated affection of the temperature sense, and even anæsthesia for heat combined with conservation of the sense of cold has been described by Chatin.[3] Ferenczi and Schaffer had an opportunity of observing anæsthesia for heat combined with hyperæsthesia for cold, which was felt to be painful (psychrohyperæsthesia). In most of my cases the *sense of locality* was specially impaired, and even to such a degree that a prick in the hand was localised in the face. The anæsthesia may be associated with a peculiar kind of hyperæsthesia, in which painless and even simple tactile stimuli produce a "queer unpleasant" sensation, or if the stimulus is more intense, a persistent and very acutely painful sensation.

Liepmann (*N. C.*, 1904) found in one case that the sense of pain was abolished in the deep parts although it was retained by the skin. Lewandowsky (*D. m. W.*, 1907) described a feeling of cold which occurred spasmodically in the affected side of the body.

A case of Schaffer's proves that bilateral foci may cause bilateral hemianæsthesia, *i.e.* an anæsthesia of the whole body, whilst in unilateral foci sensory disorders have been observed, though not with certainty, on the same side of the body (Faure, Müller). The view that the

[1] *Orvosi Hetilap.*, 1902. [2] *N. C.*, 1905. [3] *Rev. de Méd.*, 1900.

sensory centres of each hemisphere are related to both sides of the body (Monakow, Müller, Hoppe) is in any case true only to a limited extent.

I do not agree with the statement of Dejerine and his pupil, Long, that hemianæsthesia is always associated with hemiplegia. In the most marked cases of this kind which I have had an opportunity of examining, the paralytic symptoms were either absent or merely indicated (in the leg, for example), whilst *hemiataxia* was frequently combined with the hemianæsthesia. On the other hand there may be hemiplegia without any affection of sensibility, although very probably it is often accompanied at its first onset by slight disturbances of sensibility (as we see from the investigations of Brécy [1] and Gordon,[2] and as Marino [3] maintains).

Symptoms of sensory irritation—apart from pain due to muscular tension—occasionally appear in affections of the sensory conduction tract (less often in cortical affections). These take the form of *violent pain* in the opposite side of the body. This is apparently due to lesions which do not cause complete interruption of the sensory tract. Thus a focus of softening in the optic thalamus, which merely touched the tract, produced severe pain and hyperæsthesia (Edinger).[4] Interesting cases of this kind have also been published by Greiff,[5] Mann, Biernacki, Reichenberg, Oliver-Williamson, Schupfer, Klippel, Touche, Dejerine, Thomas, Roussy, and others. Anton [6] and Bechterew have studied this *hemialgia*, *monalgia*, etc.; the latter points out that the corresponding sensations may be mingled with thermic sensations (thermalgia, etc.). Notwithstanding the unilateral pains and paræsthesiæ, objective sensibility may be entirely unaffected, as I have sometimes found, and as Marie and Guillain have noted.

The close relation of the sensory to the *optic tract* explains the frequent combination of hemianæsthesia with *hemianopsia*. Thus, to give only one example, I have had under my care a case of gunshot wound of the internal capsule, in which there was at first severe pain in the opposite side of the body and later hemiparesis, hemihypæsthesia (partial sensory paralysis for *heat* and *cold* in the leg, although pain was still produced by heat, etc.), and hemianopsia. It is true, however, that hemianopsia is often absent both in focal diseases of the internal capsule and in those of the optic thalamus.

Some cases have been observed (Türck, etc.) in which the hemianæsthesia is associated with diminution or *loss of taste*, *smell*, and *hearing* on the same side, and with amblyopia and concentric narrowing of the field of vision. Charcot, however, assumes that the sensory conduction tract contains fibres which pass from the sensory nerves to the cortex, and more especially a fasciculus which conveys visual impressions to a hypothetical visual centre for the retina of the opposite eye. Although it is certainly the case that the optic and acoustic nerve tracts lie very close to the sensory tract, it is nevertheless the fact that injury of the former causes hemianopsia, whilst unilateral lesion of the latter seems to give rise to no *persistent* functional disorder. Whenever,

[1] *Thèse de Paris*, 1902.

[2] *Journ. Nerv. and Ment. Dis.*, 1903.

[3] *Ann. di Neurol.*, 1904. Sterling states that the perception of vibration is diminished in hemiplegia, and Sandberg (*Z. f. N.*, xxx.) finds that diminution of the sense of pressure and the power of localisation is particularly common.

[4] *Z. f. N.*, i.

[5] *A. f. P.*, xiv.

[6] *Prag. med. Woch.*, 1899

therefore, so-called mixed or sensory hemianæsthesia is met with, the case is one of hysteria, or of a combination of organic disease and hysteria or some other functional neurosis. This is shown by the fact that in some cases of this kind the sensory disorder was transferred or cured by the use of a magnet (Vulpian, Bernheim, Oppenheim).

Uhthoff (" Bericht der xxx. ophthal. Gesellsch.," Wiesbaden, 1903) gives the same explanation of his interesting case.

It is certainly conceivable that in hemiplegia—whether associated with hemianæsthesia or not—an hysterical hemianæsthesia may be produced by autosuggestion, as Bernheim has again recently pointed out (*Rev. de Med.*, 1902). One might also imagine that a cicatrix in the sensory path might have a similar effect, like the scar from a wound in an extremity (see section on traumatic neuroses).

Bechterew [1] (with whom Higier and others agree) has pointed out another possibility which deserves consideration, namely, that vasomotor tracts for the opposite side of the body may pass through the " carrefour sensitif " or its neighbourhood, and that injury of these tracts may affect the circulation in the peripheral sensory organs and thus impair their function. In this way a focus in the internal capsule may produce a true " hysterical " hemianæsthesia. I have seen a few cases which one might explain in this way, but I have also seen others in which there were no vasomotor disorders and no indication of hysteria. In a very large majority of my cases the hemianæsthesia, although of organic origin, was *simple*, *i.e.* it was not accompanied by the sensory symptoms of which we have been speaking. As Verger and Brécy have rightly said, this hemianæsthesia is (if we except unusual conditions, such as very large foci) neither so extensive nor so intense as it tends to be in severe cases of the hysterical form.

Diseases which are limited to the corpus striatum or the lenticular nucleus, without injuring the internal capsule either directly or by pressure, do not necessarily give rise to any appreciable sensory disorder. In lesions of the optic thalamus it depends on the special site of the focus whether sensory symptoms appear or not. Dejerine and his pupils (Thomas, Roussy,[2] see p. 642) regard these unilateral sensory disorders chiefly as symptoms of affection of the thalamus and indeed mainly of the external thalamic nucleus and its neighbourhood. Amongst the symptoms of lesion in this region they include : 1. slight, usually flaccid, and very transient hemiplegia (without Babinski's sign, etc.) ; 2. persistent hemianæsthesia, especially bathyanæsthesia, and sometimes also hyperæsthesia ; 3. severe and inveterate pain in the hemianæsthetic side ; 4. slight hemiataxia ; 5. hemichorea or hemiathetosis ; 6. occasional bladder disorders, etc. (see p. 651). They think hemianopsia an unusual symptom. There was no paralysis of the mimic facial movements in their cases. Touche found anæsthesia also in focal diseases of the external capsule, and in particular of its posterior segment, which is contiguous to the island of Reil and the temporal region, but further investigation is required to prove that this region is intimately concerned in sensibility (Marie). Affections of the ventral portion of the crus cerebri do not cause any impairment of sensibility. If, however, the *tegmentum* and the region of the fillet be affected here, in the area of

[1] *N. C.*, 1894.

[2] See his comprehensive monograph, " La couche optique," etc., Paris, 1907.

the corpora quadrigemina, or in the pons and medulla oblongata, there is hemianæsthesia of the opposite side without involvement of the sensory organs. The auditory nerve of the opposite (Henschen) or of both sides (Siebenmann [1]) may be alone affected.[2] It has not yet been definitely ascertained which portion of the tegmentum must be destroyed in order to produce sensory disorders. The fillet and the formatio reticularis are chiefly concerned (Kahler and Pick, Henschen, Moeli), the former being in any case the most important portion of the sensory path. Unilateral affections of the pons and medulla oblongata, which involve the fifth nucleus or the roots, may give rise to *hemianæsthesia cruciata* or *alternans*, *i.e.* anæsthesia of the same side of the face and of the other part of the opposite side of the body. This hemianæsthesia is as a rule dissociated, *i.e.* it involves only the senses of pain and temperature. Affections of the medulla oblongata (*q.v.*) may produce a combination of unilateral bathyanæsthesia and ataxia with crossed anæsthesia to pain and temperature (Oppenheim, Hun,[3] Wallenberg, Breuer-Marburg,[4] and others). In one of my cases the development of *a hemihyperæsthesia alternans*, *i.e.* hyperæsthesia of one side of the face and of the opposite side of the body, was the first sign of involvement of the medulla oblongata.

In another case a focal lesion of the medulla oblongata produced, in addition to right recurrent paralysis, left hemianalgesia and thermanæsthesia and also hyperæsthesia of the right side of the body, which was shown by the fact that the patient, who was a merchant, could not judge of different cloths by feeling them, as the touch gave rise to an unpleasant, painful sensation.

E. Müller (*Z. f. N.*, xxxi.) agrees with Wallenberg and others, that a kind of Brown-Séquard unilateral paralysis may develop even in the area of the trigeminus, as the sense of contact and of deep pressure may be diminished in one-half of the face and the sense of pain and temperature in the other. He has moreover found in a case of hemianæsthesia alternans that the patient on the thermanæsthetic side of his body felt temperatures of 28-29° as normal, *i.e.* as mild, and all the higher degrees as colder and the lower degrees as warmer than on the unaffected side, so that he experienced a constant sensation of warmth on the affected side.

In the lower segments of the medulla oblongata, lesions of the interolivary layer produce sensory anomalies, but these conditions are not yet fully explained (see p. 640 and the chapter on diseases of the pons and medulla oblongata).

Affections of the sensory centres and tracts may cause *ataxia* in the extremities of the opposite side of the body. *Hemiataxia* has been observed in diseases of the parietal lobe, the motor centres (?), or the posterior central convolution, the internal capsule, the corpora quadrigemina, and the pons. Whether the tracts concerned are those interruption of which gives rise to anæsthesia or other tracts, we do not as yet definitely know. The hemiataxia is certainly most marked in cases in which a focal disease in the posterior limb of the internal capsule or in the thalamo-cortical tract has produced hemianæsthesia without hemiplegia, and I have no doubt that lesion of the tracts and centres for the sensibility of the deep parts may give rise to hemiataxia.

[1] *Z. f. Ohr.*, 1896.

[2] In one case under my care, an apoplectic attack with hemiplegia was followed by the development of a *diplacusis* (the patient hearing the key-note with the addition of the third).

[3] *N. Y. Med. Journ.*, 1897.

[4] *Obersteiner*, ix. See also Rossolymo, *Z. f. N.*, xxiii.; Henschen, *N. C.*, 1906; Babinski-Nageotte, *R. n.*, 1902; L. R. Müller, *A. f. kl. M.*, Bd. lxxxvi.; E. Müller, *N. C.*, 1906; *Z. f. N.*, xxxi.; Kutner-Kramer, *A. f. P.*, Bd. xlii.; Wallenberg, *Z. f. N.*, xxvii.; Babinski, *R. n.*, 1906.

According to Dejerine and Egger (*R. n.*, 1903) the ataxia will, under those conditions, be slight (especially as compared with tabetic ataxia), but this has not been my experience.

The occurrence of *hemiataxia without any affection of the sensibility*, due to focal lesions in the basal ganglia, especially in the corpus striatum, has been described by Claparède (*Genf.*, 1897, and *R. n.*, 1903), and attributed to lesion of the centripetal paths which convey unconscious impressions to the basal ganglia. This form must, however, as far as our experience goes, be very rare in diseases of the cerebral hemisphere.

In pontine diseases associated with ataxia, the median ventral portions of the tegmentum were specially affected (Moeli [1]). In one case of lesion of the pons I have found ataxia limited to the arms.

Ataxia also occurs in lesions of the cerebellar peduncle and the cerebellum. This cerebellar ataxia is a motor symptom, and is never due to disorders of conscious sensibility. It is, moreover, homolateral. We shall discuss this question in more detail further on.

Bechterew (*N. C.*, 1905) describes, under the name of *pseudomelia parœsthetica*, a sensation as if the extremity were in another position, *e.g.* it is felt to be flexed when it is actually extended, or the paralysed extremity is felt to be constantly moving. He compares this symptom with sensory hallucinations after amputations. It does not seem to have any definite localising importance.

Vasomotor and Trophic Symptoms.—These hardly ever appear alone, and are as a rule associated with hemiplegia and hemianæsthesia. Some experience would indicate that these symptoms and hemianæsthesia are very closely related. In diseases of the motor centres which produce monoplegia, increase or diminution of the temperature of the skin of the paralysed extremity, especially of the hand, is often observed, and it is accompanied by redness, cyanosis, and frequently by œdema. Similar symptoms have been noted in surgical operations in this region. I have once seen, after an exploratory trephining of the motor zone with incision and puncture, a tendency to flushing and hyperæmia of the opposite side of the face and to tachycardia. I have also had some cases under my care which I could only diagnose as the *vasomotor form of Jacksonian epilepsy* and vasomotor monoplegia; the symptoms were attacks of vasomotor disturbance in the arm and face of one side, with or without loss of consciousness, and persistent vasomotor and oculo-pupillary disorders in the same region, with only slight paresis and atrophy (the latter probably also of vasomotor origin). The fact that the brain cortex contains vasomotor centres (in the neighbourhood of the motor) has been corroborated by experiment (see p. 625). An opportunity of ascertaining the presence of symptoms of this kind is more frequently afforded by diseases of the conduction tracts, and by the ordinary capsular hemiplegia or hemianæsthesia. It is probable that the vasomotor nerve path, hitherto undefined, ends in the neighbourhood of the sensory tract. Parhon-Goldstein,[2] on the other hand, localise it in the anterior limb of the internal capsule. Lesion of the central ganglia has also been regarded as a cause of vasomotor and trophic symptoms (see p. 651). There is no doubt that vasomotor disorders may also be due to disease of the medulla oblongata. In the ordinary form of hemiplegia, the œdema is as a rule limited to the paralysed limb, and when nephritis is present, the œdema may be confined to this side. Loeper and Crouzon [3] have recently contributed to the study of this question.

[1] *A. f. P.*, xxiv. [2] *Roum. méd*, 1899, and *R. n.*, 1902. [3] *Nouv. Icon.*, xvii.

Diminution of the blood pressure in the arteries of the affected side (Féré,[1] Tissier), and vaso-dilatation of the capillaries with capillary pulsation, have also been observed (Marie and Guillain).[2] Subcutaneous and muscular hæmorrhage have been included by Parhon and Goldstein among the symptoms. They have also found that the sweat secretion was increased on the hemiplegic side when pilocarpin was injected. A tendency to bed-sores was noted in some cases, especially in severe hemiplegia. Other "trophic" symptoms, such as eruptions of pemphigoid blisters, sometimes appear on the skin. I have only once seen the development in a case of hemiplegia and hemianæsthesia of ulcers on the fingers, which were so persistent as to necessitate removal of the phalanges. I have seen the same thing occur in one case of infantile spastic hemiplegia. An extremely rare symptom is that of the hair turning grey on one side (Brissaud). Steinert mentions loss of hair and hypertrophy of the subcutaneous fatty tissue. Gangrene of the paralysed limb has once been noted (Preobrajenski), but the connection is not quite clear.

I have seen a combination of hemianæsthesia and neuroparalytic keratitis in the eye of the same side. Although the form of the hemianæsthesia indicated a focus in the internal capsule, I do not venture to attribute the keratitis also to this origin, and I must in the meantime leave the unusual condition unexplained.

The *joints* of the affected limb are often diseased. The changes are partly of an accessory nature and are due to the inactivity and permanent fixation of the extremity. They may, however, originate in another way: A short time (one to four weeks) after the onset of the hemiplegia, or even at a later stage, when a certain amount of movement has been regained, there is an acute or subacute development of synovitic arthritis, with redness and usually with slight swelling of the joints. Hæmorrhages very seldom occur at the same time. It is exceedingly probable that in such cases the influences at work are mainly of a trophic nature. We have already alluded to the combination of joint affection and muscular atrophy in hemiplegics.

Visual Disorders

The most of the literature of this section will be found in Uhthoff, Graefe-Saemisch "Handbuch," 2nd edition; in Wilbrand-Saenger, *Neurol. d. Auges*, Bd. iii., and in Schmidt-Rimpler, 'Nothnagel," Bd. xxi., 2nd edition, 1905.

It is hardly possible to distinguish conditions arising from lesions of the optic nerve from those of cerebral origin, and the fact that the optic nerve is genetically a part of the brain is a further justification for discussing its affections at this point. The optic nerve is, in fact, the part which comes to the periphery and is therefore accessible to direct examination. The importance of ophthalmoscopic examination in the diagnosis of brain diseases hardly requires to be insisted upon. To neglect this step in making the diagnosis of a brain disease is simply to disregard the one loophole through which we may gaze into a region full of mystery.

Affections of the optic nerve may be recognised partly by *ophthalmoscopic* examination and partly by testing the sight, or by a combination of both these methods.

[1] *Soc. de Biol.*, 1893. [2] *R. n.*, 1902.

The changes which, in addition to errors of development, are revealed by the ophthalmoscope are *optic neuritis* or *choked disc*, and *atrophy*, of which there are various forms.

Between optic neuritis and choked disc, there is merely a difference of degree. We generally speak of choked disc when there is a distinct prominence of at least 2-3 mm. (a difference of refraction of 2 dioptres). The condition is characterised by blurring and swelling of the disc, and by the want of definition of its margin. If the condition is marked, the disc is very *prominent*, is reddish or grey-red in colour, cloudy and

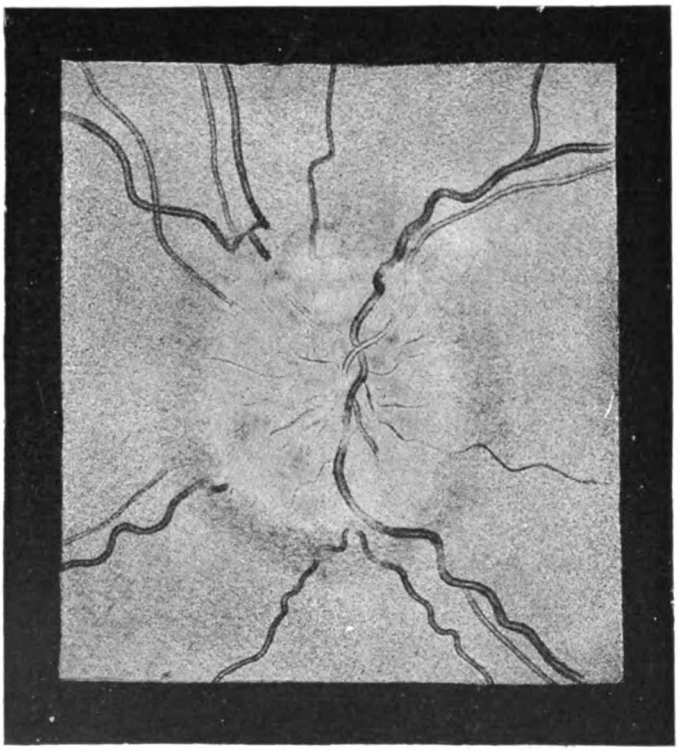

FIG. 300.—Optic neuritis (or choked disc) in brain tumour. (After Gowers.)

blurred, and its margins are indistinct, the veins dilated, the arteries contracted; the vessels, which are in some parts invisible, appear to break off at the margins of the papilla. The diameter of the disc may be three times as large as normal. Hæmorrhages and white spots (fatty degeneration) also appear (Fig. 300). It is not, however, always easy to draw the dividing line between optic neuritis and choked disc. Many writers are inclined to regard inflammatory changes in the papilla as quite apart from choked disc.

Choked disc may be confused with *albuminuric retinitis* limited to the papilla. There are also *congenital malformations* (usually associated with hypermetropia) which may closely resemble optic neuritis. This affection, known as congenital *pseudoneuritis optica* (Wecker, Bristowe,

Uhthoff, Nottbeck,[1] L. Pick [2]), is chiefly distinguished from choked disc by its unvarying character.

The *sight* is often quite normal in choked disc. In advanced cases it is usually impaired, and there is irregular contraction of the field of vision and diminution of the central acuity of vision. This diminution of function may increase into complete blindness. Repeated attacks of sudden and transient amaurosis have been observed in choked disc. This is probably the result of periodic increase of the brain pressure (in cerebral tumours, etc)., which gives rise to compression of the optic chiasma by exudation of fluid into the third ventricle.

Brain tumour is the most common and the most important cause of optic neuritis or choked disc. When this is the cause the neuritis is almost always bilateral, although it may at first be limited to one eye. Its occurrence has practically no relation to the site of the neoplasm (see chapter on brain tumour). We can merely refer at this point to the occurrence of choked disc in so-called pseudo-tumour cerebri and to Reichhardt's acute brain swelling.

Internal hydrocephalus or serous meningitis (*q.v.*) not infrequently gives rise to choked disc. Cases of this kind have greatly multiplied during the last ten years, so that we have every reason to assume a connection between the two conditions, especially when bilateral optic neuritis develops in an adult, acutely, subacutely, or even in a chronic form, along with other symptoms of increased intracerebral pressure. In such cases I have often seen the patient become quite suddenly blind, *e.g.* after bending or moving the head abruptly. Amaurosis is a common symptom also in *brain abscess* and sinus thrombosis. Optic neuritis is not so frequent in acute non-suppurative *encephalitis*, but it is common in cerebral syphilis and in the various forms of (basal) *meningitis*. I have only once found choked disc in chronic recurrent hæmorrhage. Other cases of this kind have been reported (Mackenzie, Michel, Priestley Smith), but these have apparently been for the most part instances of rupture towards the base.

Nonne (*M. m. W.*, 1906; *N.C.*, 1906) has seen a few cases in which the choked disc in cerebral hæmorrhage could only be attributed to the general increase of brain pressure. Lewandowsky has also seen this (a verbal communication).

Choked disc is more often observed in *hæmorrhagic pachymeningitis* and in meningeal hæmorrhages of traumatic origin, with or without fracture of the optic foramen, and it then takes the form of " hæmatoma of the optic nerve sheaths " (Michel, Bergmann, Panas, Förster, Uhthoff, Fleming, Liebrecht, Halbey, etc.). Blood may also find its way into the optic nerve sheaths from ruptured aneurisms of the basal arteries.

Choked disc has often been observed in contusions of the skull, as in the case of Bachauer (*D. m. W.*, 1904). For an exhaustive presentation of this subject Wilbrand-Saenger should be consulted (*loc. cit.*). Retinal hæmorrhages are by no means uncommon in basal fractures, as I myself have seen and Fleming has described (*Edin. Med. Journ.*, 1903).

Uhthoff thinks it very doubtful whether optic neuritis ever occurs in thrombosis and embolism of the cerebral arteries. In any case this is extremely rare, and a typical choked disc is hardly compatible with this diagnosis (unless nephritis be present).

[1] *Inaug. Diss.*, Marburg, 1897.

[2] *Z. f. Aug.*, 1904.

Zacher has described a case (*N. C.*, 1901) in which optic neuritis was associated with a bilateral focus of softening in the frontal lobes. Trömner (*N. C.*, 1906) has also found choked disc in a patient suffering from cerebral softening.

Uhthoff discusses the ocular symptoms of thrombosis of the cerebral sinus in the *M. f. P.*, xxii.

There can be no doubt that there is a *rheumatic* form of optic neuritis, although it is probably due to some *infective agent*. Its association with "traumatic neuritis" of other cranial nerves, *e.g.* the facial, also points to this origin. At all events optic neuritis may develop from acute infective diseases, typhoid, scarlatina, influenza, erysipelas, etc. Uhthoff has, it is true, attributed only a few of his cases to this cause, but a great number of such observations may be collected from the literature. As regards the occurrence of optic neuritis and other affections of the optic nerve in *syphilis*, the chapter on this subject should be consulted.

Neuritis or neuro-retinitis is in rare cases due to *gout*, as Hirsch, for example, has observed.

I have repeatedly found that optic neuritis and choked disc may develop in *chronic lead poisoning*, as well as other forms of visual disorder (hemianopsia, amaurosis of a uræmic nature, transitory amaurosis without nephritis (?), etc.). Elschnig (*W. m. W.*, 1898) has studied this subject very fully.

It is a very remarkable fact that optic neuritis may be due to *chlorosis*.

Cases of this kind have been published by Thomson (*Brit. Med. Journ.*, 1894), Burton-Fanny (*Brit. Med. Journ.*, 1894), Jolly (*ibid.*), Patrick (*Journ. Nerv. and Ment. Dis.*, xxv.), Muntendam (*Weekbl. van het Nederl.*, 1900), Hawthorne (*Brit. Med. Journ.*, 1902), Evans (*Lancet*, 1904), and others.

One writer has lately collected twenty such cases from the literature. Some of the visual disturbances observed in anchylostomum duodenale (Nieden,[1] Inouye [2]) may belong to this class. In one anæmic patient who suffered from menorrhagia and from very severe headache during the menses, I found an optic neuritis which suggested some grave condition; the further course of her illness and her spontaneous recovery showed, however, that the neuritis had developed under the influence of the anæmia and of the circulatory changes associated with the period. I imagined that a serous meningitis caused by the anæmia or hydræmia, and increased by the effect of menstruation had given rise to a slight degree of neuritis.

It is very seldom present in ulcerative endocarditis (Mackenzie, Broadbent). It may occur in malformations of the skull, such as the so-called tower-shaped skull.

Cases of this kind have been recorded by Michel (*A. f. Heilk.*, 1873), Enslin (*Graefes A.*, Bd. lviii.), Weiss-Brugger (*A. f. Aug.*, Bd. xxviii.), Velhagen (*M. m. W.*, 1904), Alexander (*D. m. W.*, 1903), Oberwarth (*A. f. Kind.*, Bd. xlii.), and Uhthoff has discussed fully the mode of production of tower-shaped skull and its effect upon the eye (*Vorträge*, Breslau, 1904). Meltzer (*B. k. W.*, 1908) thinks that the tower-shaped skull is produced by serous meningitis or hydrocephalus, and that synostosis is the result of a special reaction of a rachitic skull to this process.

It is doubtful whether arteriosclerosis may produce a true optic neuritis. We [3] have had to accept this as the explanation of a few of our cases.

[1] *D. m. W.*, 1903. [2] "La clin. ophthalm.." 1902.

[3] Oppenheim-Siemerling, *Charité-Annalen*, xii.

Otto has found simple diminution in the size of the nerve fibres as the most common condition. Liebrecht regards the pressure atrophy as the primary process, and thinks this may be followed by proliferation of connective tissue and new formation of vessels.

See also Bernheimer, *Graefes Arch.*, Bd. xxxvii. Stölting (*Kl. M. f. Aug.*, 1905) has discussed the diseases of the optic nerve which are due to atheroma of the arteries. The investigation of Elliot Smith (*R. of N.*, 1905) is a practical confirmation of the knowledge already derived from experience.

It is well known that the optic nerve and the retina may be affected, and that visual disturbances may be caused by diseases of their vessels, especially of the central artery of the retina. Embolism of the central artery is the most common lesion, but this vessel may also show arteriosclerotic changes and local thrombosis. Michel distinguishes between arteriosclerotic, marantic, and compression thrombosis of the central retinal artery. Thrombosis of the vena centralis retinæ has also been described (Clermont, etc.). See also Gonin (*Arch. d'Ophth.*, 1903), Harms (*Graefes Arch.*, Bd. lxi.), Péchin (*R. n.*, 1906). We cannot here discuss such unusual processes as septic retinitis, the amaurosis which follows injection of paraffin into the region of the eye, or the relation of retinal hæmorrhage to brain disease, etc.

I have in two cases seen an optic neuritis develop in persons who showed a marked tendency to vasomotor disorders (local syncope, etc.), and I could discover no other cause or explanation. I quote these cases, however, with all reserve.

The *origin of choked disc*—a term invented by Von Graefe—is as yet insufficiently explained. Two main opposing theories must be considered. According to one (Schmidt-Rimpler, Manz) the explanation is that the cerebro-spinal fluid is forced by the increased pressure into the optic nerve sheath, where it presses so severely upon the veins as to cause actual congestion, and œdema, which may be followed by inflammation. According to the other theory (Leber, Deutschmann,[1] Elschnig [2]), toxic products are directly responsible for inflammation of the discs, optic neuritis, or papillitis.

Recent observations (Bruns, Oppenheim, Saenger,[3] Kampferstein,[4] Liebrecht,[5] Uhthoff,[6] Finsch, Paton,[7] Krüdener,[8] Taylor,[9] Spiller-Frazier [10]) point almost unanimously to a mechanical origin of choked disc, as the condition in very many cases rapidly disappears after decompressive trephining. We cannot here consider the other theories of Adamkiewicz, Parinaud, Jackson, Sourdille, etc. Duret has treated the matter very thoroughly in his work on brain tumours.

Choked disc may entirely disappear, *e.g.* in brain syphilis or successfully treated brain tumours. Opening of the skull in inoperable tumours of the brain may in itself suffice to cure choked disc. In exceptional cases the affection of the optic nerve may recover spontaneously, although the brain disease persists. If the causal disease persists a long time, the choked disc may gradually pass into *atrophy* of the optic nerve.

Atrophy of the optic nerve may therefore be of a *secondary* nature, *i.e.* it may be preceded by optic neuritis or it may be due to compression of the optic nerve at a site more or less behind the eyeball, or it may be *primary*.

Primary atrophy of the optic nerve, which is almost always *bilateral*,

[1] "Über Neuritis optica," Jena, 1894.
[2] *Graefes Arch.*, Bd. xli.; *N. C.*, 1894 and 1902.
[3] *Wien. m. W.*, 1904; *N. C.*, 1905.
[4] *Kl. M. f. Aug.*, Bd. xlii.
[5] *N. C.*, 1904.
[6] *N. C.*, 1904.
[7] *R. of N.*, 1905.
[8] *Graefes Arch.*, Bd. lxv.
[9] *Ophthalm. Soc. Trans.*, xiv.
[10] Tumour of the Cerebrum," Phila., 1906.

is specially common in tabes dorsalis and paralytic dementia; it is possibly in very rare cases a direct symptom of syphilis, and apparently in a few cases it may be an independent affection. The latter is, however, so unusual that when atrophy of the optic nerves is the only symptom, a provisional diagnosis of tabes dorsalis (or paralytic dementia) should be given, as this symptom may precede the others for ten years or more. The atrophy may be recognised from the evident pallor, the whiteness or absence of any colour in the discs, their margin standing out with abnormal distinctness. We should remember, however, that even in the normal eyes there are marked individual differences in the colouring and vascularity of the discs, and also that the central part is as a rule paler than the peripheral. The ophthalmoscopic diagnosis of commencing atrophy must be made with great care, and continued observation and repeated careful testing of the sight is usually necessary.

A very remarkable phenomenon, to which I have already referred in another connection, has been described to me by the patients in four cases of atrophy of the optic nerve. Although they were blind, they had in regular alternation the sensation, one day, of a bright light, and the next day of a dark shadow before their eyes.

Secondary atrophy, due to compression or injury of one optic nerve (tumours at the base of the skull or in the orbit, gunshot wounds, basal fractures extending into the optic foramen, carious processes, etc.), gives rise to unilateral affection of vision, which may develop into blindness In this descending atrophy the visual disturbance precedes the ophthalmoscopic change.

In *multiple sclerosis* (*q.v.*) atrophy of the optic nerve is almost always partial, and very seldom becomes complete. The visual disorder therefore hardly ever becomes permanent and complete blindness. • Although it may also have a neuritic origin, marked optic neuritis is not often observed in disseminated sclerosis; it may appear in a transient form and usually at the commencement of the disease. I have only twice seen a typical unilateral optic neuritis develop in the course of disseminated sclerosis, and in these cases it disappeared within a short time, whilst in a comparatively large number of our cases the existing disturbance of vision or partial atrophy of the optic nerve could only be attributed to a neuritis which had preceded—sometimes by even ten years—the onset of the other symptoms (see pp. 335 and 338). Bruns and Nonne have both observed choked disc in disseminated sclerosis.

Retrobulbar neuritis is usually, though not always, of toxic origin, and it gives rise to symptoms of toxic amblyopia. This form is most apt to develop in the course of polyneuritis. It may also occur after acute infective diseases such as influenza and diphtheria, and in the course of myelitis (*q.v.*).

Chronic *alcoholism* is the most common cause of *toxic amblyopia*, and nicotine poisoning the next. It is seldom due to bisulphide of carbon, quinine, iodoform, Indian hemp, etc., and there is still some doubt as to whether it may really be produced by salicylates, carbolic acid, ergot (Orloff, *Monit. russ. neurol.*, 1904), and other poisons, to which it has been ascribed in some instances. Ruault has experimentally studied the effect of quinine upon the optic nerve. I have seen not a few cases of retrobulbar neuritis from Michel's Clinic, in which no definite cause could be ascertained. Cases of disturbance of vision associated with atrophy have lately been observed after the use of extract of filix mas and of thyroidin. The injurious effect of the former

preparation upon the optic nerve has been experimentally confirmed by Masius and Mahaim (*Bull. Acad. de Méd. Belg.*, 1898). Stuelp has lately published similar cases (*A. f. Aug.*, Bd. li.). In the retrobulbar neuritis after the use of thyroidin, large doses of the drug have usually been given over a long period to corpulent persons. Coppez, for instance, saw five cases of this kind. In retrobulbar neuritis the characteristic symptom is bilateral and relatively central scotoma for red and green in a zone—an oval with long diameter horizontal—which extends outwards from the fixed point and involves both this point and the blind spot. There is sometimes a smaller scotoma for blue, and in rare cases an absolute scotoma for white. The central acuity of vision is more or less reduced. This visual affection is due to an interstitial neuritis of the *papillo-macular* bundle in the optic nerve trunk (Samelson,[1] Uhthoff,[2] and others). Sourdille and Schieck[3] think that it is mainly due to vascular processes. Nuel ("Eighth Internat. Med. Congress," Paris, 1900) and others consider the primary process to be an affection of the nerve fibres, and the interstitial changes to be secondary to these. There is also a form of retrobulbar neuritis in which the field of vision is intact at the centre, and irregularly narrowed from the periphery inwards. The ophthalmoscopic appearances may be negative, but partial atrophy of the optic nerve usually develops, causing *pallor of the temporal halves of the discs*. There is seldom definite optic neuritis. Amongst the recent contributions to this subject we may mention the paper by Gunn (*Ophthal. Rev.*, 1905).

This affection also occurs in diabetes; that is to say, there may be a central scotoma, showing at first on ophthalmoscopic examination either no change, or merely slight neuritis, but at a later stage the signs of partial atrophy. Schmidt-Rimpler has verified this interstitial neuritis by pathological investigation. Amblyopia and atrophy of other nature is less common in diabetes. We can only mention the occurrence of diabetic retinitis. In a few cases the diabetic atrophy of the optic nerve can be traced to an endarteritis of the central artery of the retina (Leber and Hummelsheim). Retrobulbar neuritis may also be of syphilitic origin (Wilbrand). Sourdille has recently studied the pathological process in post-infective optic neuritis (*La Clin. ophthal.*, 1903). The etiology of retrobulbar neuritis of non-toxic origin is still unexplained. There is a *hereditary* form which affects several members of a family and usually appears in youth (from the twelfth to the twentieth year). In these cases there is generally an absolute central scotoma, and the affection may be limited to one side. It may begin with pain in the orbit.

Transient amblyopia, probably due to retrobulbar neuritis, has occasionally been observed during the time of lactation. Leber has described a hereditary form of atrophy of the optic nerve, and Kowalewski[4] has recently demonstrated some cases of this kind.

There are, further, hereditary family nervous diseases, which are characterised by the combination of idiocy with amaurosis and atrophy of the optic nerve (Sachs, Warren Tay, Falkenheim, Frey, and others; for the literature see the chapter on infantile cerebral paralysis). These show characteristic changes in the macula lutea. A *congenital* optic atrophy apparently sometimes occurs. Atrophy of the optic nerve is rare in the ordinary form of infantile cerebral paralysis (W. König, etc.).

The etiological relation of head injuries to optic neuritis (Adamük, Cramer, etc.) has not yet been sufficiently explained.

The subject of affections of the optic nerve caused by diseases of the nose and its accessory sinuses has been exhaustively treated by Onodi.[5]

There are but few cases recorded of the *senile* form of atrophy of the optic nerve. Through the courtesy of Dr Michel I have had an opportunity of observing a few such cases. In one long-lived family the affection appeared in several of its members during old age.

Great interest is attached to the visual disturbances which are caused by *dazzling*, by intense illumination of the retina, and especially, as

[1] *Graefes Arch.*, Bd. xxviii.
[2] *Graefes Arch.*, Bd. xxxii. and xxxiii.
[3] *Graefes Arch.*, Bd. liv.
[4] *D. m. W.*, 1906.
[5] *B. k. W.*, 1906, and "Der Sehnerv und die Nebenhöhlen der Nase," Wien, 1907.

recent experience has shown, by the effect of light from the electric arc (*electric ophthalmia*). The affection, which is usually of the nature of scotoma, develops with pain in the eye, photophobia, blepharoclonus, etc. As a rule it rapidly disappears, but it may develop into blindness. The result of ophthalmoscopic examination is negative, or it may show spots in the macular region (Uhthoff) and less often marked atrophy.

Winselmann (*Clin. ophth.*, 1903) saw these symptoms after the eyes had been dazzled by watching an eclipse of the sun, and Tower, Nelson, Laker, and others after a flash of lightning. Lundsgaard (*Hospitalstid.*, 1904) describes the injuries of the eye caused by an electrical short circuit.

Lesions of the *optic chiasma* and of the *optic tract* do not necessarily cause changes on the fundus of the eye, although when they are of long duration they may lead to the development of a descending atrophy, and new growths in this region also tend to produce optic neuritis. The *symptoms* are very characteristic. Lesions in the chiasma generally involve the middle portion which contains the decussating fibres. The functional result of this process is a *bitemporal hemianopsia* caused by *amblyopia of the inner half of each retina*. We can easily see that an extension of this process is likely to cause complete blindness, or blindness of one eye with temporal hemianopsia of the other. On the other hand nasal hemianopsia caused by a lesion limited to the external fibres of the chiasma is a very rare occurrence.

So called inferior and superior hemianopsia is also an exceptional symptom, and one which is difficult to explain. Cases of this kind have been reported by Mauthner (*Öster. Zeitschr. f. Aug.*, xviii.), Schweigger, and Nothnagel (*Wien. med. Bl.*, 1888). Salomonsohn found twelve cases reported in the literature. Russell has reported a case with an autopsy. There are also a few cases (Ole Bull, Linde) in which the hemianopsia was confined to one eye; these are difficult to interpret.

The diseases which affect the chiasma are usually new growths, mainly syphilitic (see chapter on brain syphilis), or tubercular, and other tumours of the hypophysis, etc. In hydrocephalus the middle portion of the chiasma may be directly affected by the bulging of the floor of the third ventricle, and bitemporal hemianopsia may result. It is often present in acromegaly.

Moreover, simple, circumscribed inflammatory conditions in the chiasma may in rare cases develop into atrophy (Leszynsky).

Reuchlin (*M. f. U.*, 1906) attributed the symptom in one case to a basal fracture with laceration of the chiasma.

Affections of the optic tract give rise to *bilateral homonymous hemianopsia*. The way in which this symptom is produced is shown in the diagram in Fig. 277. The diagram shows also how it may result from lesions of any part of the intracerebral visual path and centre. As a guide to local diagnosis this symptom has therefore a somewhat limited value, but the site of the lesion can usually be determined from the accessory symptoms. Lesions of the optic tract as a rule involve some of the other basal cranial nerves, as it is only in very rare cases that a lesion such as hæmorrhage, softening, inflammation, neoplasm, remains limited to the optic tract. Symptoms of focal lesions in the temporal lobe—aphasia, hallucinations of smell, anosmia (?), etc.—may be

associated with hemianopsia of this origin. Diseases of the optic thalamus or of the external geniculate body, which give rise to hemianopsia, usually involve the internal capsule and therefore cause hemiplegia, hemianæsthesia, etc., on the same side as the hemianopsia.

Lesions of the optic centre in the cerebral cortex may produce hemianopsia as the only symptom. A great number of such cases have been recorded; they are usually due to foci of softening, not infrequently to tumours, and occasionally to trauma. We shall refer later to certain peculiarities of this cortical form. Lesion of the optic radiation in the right hemisphere gives rise to practically the same symptoms as lesion of the optic centre. In the left hemisphere the optic nerve tract passes in the same way through the temporal and occipital lobes, but here it traverses other regions which are closely connected with the speech processes. Its interruption, therefore, usually causes not only right-sided hemianopsia, but also symptoms of aphasia.

In hemianopsia the line dividing the seeing from the blind half of the field of vision is usually vertical, and is so drawn that the area of direct vision falls within that of the unaffected half of the field. There are many exceptions to this rule, however. The hemianopsia is complete if the dividing line passes through the fixation point. This is the rule when the optic tract is completely interrupted, is less common in disease of the external geniculate body, and is the exception in diseases of the cerebrum (Monakow). The hemianopsia may be incomplete, either owing to the fact that corresponding segments are unaffected, or that only the inferior or the upper quadrants of the homonymous halves of the field of vison have been involved (quadrant-hemianopsia). These partial defects of the field of vision are very seldom due to disease of the optic tract or of the primary optic centres; as a rule they are of cortical origin or due to a lesion of the optic radiation. Cases of this kind, in addition to the reports of animal experiments by Munk and Hitzig, have been described by Henschen, Pick, Bruns, Cramer-Thiem (*Z. f. Aug.*, vii.), Beevor-Collier (*Br.*, 1904), and Oppenheim. The fibres belonging to each quadrant of the retina appear to run together in the optic radiation. We cannot, however, use this symptom to determine the site of the lesion. It has, for instance been found by Henschen and others in a lesion affecting the external geniculate body.

Wehrli (*A. f. Ophth.*, Bd. lxii.) draws attention to the fact that, in the published cases of homonymous hemianopsia, the lesion was never absolutely limited to the cortex of the vision centre, the centrum ovale surrounding the optic radiation being always involved, and he therefore urges that no definite conclusions as to the exact localisation should be drawn from these cases.

The patient is usually conscious of the hemianopsia, especially if he is intelligent. He is apt to run up against things, and he tries to avoid this by turning his head towards the side upon which he is blind. Deviation of the head and eyes towards the unaffected side in hemianopsia (see above) only occurs after an apoplectic fit, and as a rule it is of short duration. In one of our cases, in which a right-sided hemianopsia was associated with paralysis of the right abducens, these two affections influenced and compensated each other in such a way that the complaints of the patient gave no indication of their existence. Right hemianopsia makes reading difficult but not impossible. One man whom I treated for this condition had great trouble in reading words, but none whatever in reading figures, even in many columns. He had formerly been an accountant.

Disturbances of orientation and of deep localisation (Exner, Pick, Anton, Hartmann), etc., have been observed in hemianopsia. These have been attributed to the special localisation of the process in the angular gyrus or on the basal surface of the occipital lobe, but the hemianopsia in itself seems to give rise to a certain impairment of the

power of orientation (see below). Thus it has been found by Liepmann and Calmus, and by Löser, that a patient suffering from right hemianopsia will, on trying to divide a line into two, make the corresponding right half too small. The disturbances of orientation are most marked when the optic sphere is affected on both sides (see below and also p. 630 and p. 624).

Hemianopsia is sometimes associated with *concentric narrowing of the field of vision*, the opposite eye being most often affected. It should always be borne in mind, however, that it is the temporal, and therefore the larger half of the field of vision which becomes blind in the opposite eye, and that a general reduction may thus be easily simulated. If the hemianopsia is associated with true concentric narrowing of the field of vision, the case is in all probability one of combined organic and functional disease, such as Uhthoff[1] has diagnosed in one of the very interesting cases which he describes. We must not, however, forget to mention that concentric narrowing of the field of vision is said to be one of the symptoms of arteriosclerosis of the vessels of the brain (Vogt).[2]

I have repeatedly found, as I have stated in earlier editions of this book, and in the general part (see p. 75), that patients in whom the ordinary tests have not indicated any hemianopic symptoms, could not recognise objects passed before the right or left halves of the field of vision, or that they did not see them if other objects (a key, a knife, and so on) were simultaneously brought before the other halves of the field of vision. This seems to me to be due to an impairment of the power of attention, but the symptom may precede the development of a true hemianopsia or it may for a long time be the only one present.

In a few cases of focal disease of the brain, instead of hemianopsia there is an amblyopia with concentric narrowing of the visual field in the opposite eye and a slight narrowing of the field of vision in the eye on the side of the lesion. This symptom has led to the assumption of a second, higher visual centre, which is connected with the whole of the retina of the opposite eye, but which also receives visual impressions from the retina of the eye of the same side. An attempt has been made to localise this questionable optic centre in the lower parietal lobe. The theory, however, stands on very slender foundations, and it is exceedingly improbable that there is any visual centre except the one in the occipital lobe.

Hemianopsia may also be incomplete in so far that it is the *colour sense* alone that is destroyed whilst the senses of light and space are intact. *Hemichromatopsia* or hemiachromatopsia seldom occurs, as Eperon, Henschen, and I have stated. The recent works of Becke (*Z. f. Aug.*, xi.) and Lenz (*Inaug. Diss.*, Breslau, 1905) show, however, that it is not so uncommon a symptom as we had thought. According to Wilbrand's theory, these various perceptions are related to different layers of the optic centre, colour being perceived in the most superficial layer. Hemichromatopsia would therefore point to lesion of the optic centre affecting the outermost marginal layers. This view is, however, rightly disputed (Vialet, Marchand). One of the two cases of this kind which I have seen could be interpreted by Wilbrand's theory. After injury of the left occiput, which had caused a deep depression, a right homonymous bilateral hemichromatopsia developed without any other focal symptoms. In another case, however, the hemiachromatopsia, which developed acutely, was associated with hemianæsthesia or hemihypæsthesia, a combination which could be most easily explained by a lesion of the posterior thalamic region or the external geniculate body, and of the adjacent area of the internal capsule. It is not impossible, however, that there was a superficial disease of the occipital and parietal lobes. This question can only be determined by further observations and pathological reports.

Lewandowsky (*B. k. W.*, 1907) has described a case which is difficult to explain. The lesion, affecting probably the left occipital lobe, had apparently destroyed the associative connection between the colour sense and the other optical ideas, the patient having lost all remembrance of the colours of most familiar objects. An interesting case of hemianopsia with blindness for red and green in the conserved field of vision is described by K. Abraham (*N. C.*, 1904).

[1] "Bericht der Ophthalm. Gesellsch." xxx., Wiesbaden, 1903.

[2] *N. C.*, 1902.

Wernicke [1] has drawn attention to the fact that the condition of the pupillary reaction is an aid to the localisation of hemianopsia, as a hemianopsia caused by disease of the optic tract gives rise to immobility of the pupil when the amblyopic half of the retina is illuminated, whilst the light reaction remains normal when the lesion of the optic tract is situated behind the corpora quadrigemina and the lateral geniculate body, or behind the reflex centre, chiefly therefore in cortical affections of vision. This view is theoretically correct, but in practice the existence of *hemianopic immobility of the pupil* is very seldom proved beyond dispute. Its occurrence has recently been confirmed by H. Salomonsohn [2] from his own and other observations. Monakow, Linde, Dercum,[3] and notably Vossius,[4] have found it present beyond a doubt.

I was able in one case to satisfy myself that this symptom was present in a patient shown to me by Loeser.

The case of Josserand (*Lyon méd.*, 1902) in which the pupillary light reflex was abolished in central blindness from bilateral softening of the occipital lobe, is still unexplained.

We have no definite knowledge as to the condition of this reflex in hemianopsia caused by disease of the primary optic centres (Henschen). Ferrier has experimentally produced hemianopic immobility of pupils in the ape.

The hemianopsia due to lesions of the cortex and the subcortical optic fibres is sometimes associated with phenomena, such as unilateral *visual hallucinations*, which are assumed to be symptoms of irritation (Seguin, Uhthoff, Seglas,[5] Erbslöh, Oppenheim, etc). These may apparently be the only symptom of an irritative lesion of the optic centre, and at all events they may precede the development of the hemianopsia. According to Wilbrand and Henschen, visual hallucinations are very common in subcortical hemianopsia. Uhthoff has shown that they are often due to disease of the eye itself, which gives rise to endoptic perceptions.

Bilateral hemianopsia is not always identical with blindness. In several cases, such as those described by Foerster,[6] Sachs,[7] Gaupp, Laqueur-Schmidt,[8] O. Meyer,[9] Touche, Marchand, Joukowsky,[10] and others, central sight was not abolished, but the power of orientation was always severely impaired. Nevertheless bilateral foci in the occipital lobe (symmetrical destruction of the cortex of the cuneus and the calcarine fissure), in the optic radiation, and perhaps also extensive destructions of the corpora quadrigemina or the geniculate body, may give rise to total blindness. In this amaurosis the pupillary light reflex is conserved if conduction is interrupted beyond the reflex centre. Henschen's view that the macula remains intact if the focus spares the calcarine fissure has been freely criticised. The case examined by Laqueur-Schmidt confirms the view of Förster and Sachs that central sight is possible if the posterior part of the floor of the calcarine fissure is unaffected.

Küstermann (*M. f. P.*, ii.) points out that paralysis of the eye muscles, which the views of some writers would lead us to expect, is absent in these cases of bilateral hemianopsia. Henschen

[1] *Fortschr. d. Med.* i.; *Z. f. kl. M.*, vi.
[2] *D. m. W.*, 1900.
[3] *Journ. Nerv. and Ment. Dis.*, 1900.
[4] *Samml. zwangl. Abh. Aug.*, iv.
[5] *Ann. méd.-psych.*, 1902. See also Becke, *Z. f. Aug.*, xi.
[6] *A. f. Ophthal.*, Bd., xxxvi.
[7] *Arb. aus d. psych. Klinik*, Breslau, 1895.
[8] *V. A.*, Bd. clviii. and clxxv.
[9] *M. f. P.*, 1900.
[10] *Nouv. Icon.*, xiv.

has observed conjugate deviation, but it should not be interpreted in this sense. Harris (*Br.*, 1897-98) states that a unilateral lesion of the cuneus of sudden onset may, as the result of an inhibiting influence of the lesion upon the centre, cause very transient total amaurosis, which rapidly develops into a homonymous hemianopsia. Bruns (*N. C.*, 1900) thinks that transient blindness is not uncommon in tumours in the occipital lobe of one side. Monakow's observation that there may be a simultaneous embolism of both occipital arteries and that cortical blindness may thus be produced by *one* attack is of great interest. The recent contributions to the subject made by Collet and Gruber (*Lyon méd.*, 1905) should also be consulted.

In addition to severe disturbances of orientation, a diminished attention to visual impressions has also been noted as a symptom of bilateral affections of the vision centres (Anton, Bischoff, Hartmann). Dejerine, Anton, and others have further pointed out that the patients are often quite unconscious of their blindness, but Redlich (*N. C.*, 1907) maintains on the other hand that such an unconsciousness is the result of a mental defect. Loss of the power of orientation or of topographical memory has been specially observed in bilateral focal diseases of the vision centre or of the optic radiation, by Förster, Lissauer, Wernicke, Reinhard, Marchand, Dide-Botcazo (*R. n.*, 1902), and others. This function is attributed by Touche (*Presse méd.*, 1901) to the fusiform gyrus, and by Dide-Botcazo to the lobus lingualis. Pick and Anton on the other hand think that the disorder is related to the angular gyrus. An interesting case of this kind is reported by Hartmann (p. 624). Niessl-Mayendorf think that disturbance of orientation is due to interruption of the commissural fibres between the two visual centres, that is to say to a lesion of the posterior portion of the corpus callosum. The symptom has been observed in rare cases of unilateral foci in the occipital region (Marie-Ferrand, Touche), and according to Lenz (*Inaug. Diss*, Breslau, 1905) it is not uncommon, especially in left-sided hemianopsia. Hartmann [1] has thoroughly studied the question.

It may be assumed that certain forms of transient blindness are caused by a *functional* or *toxic* paralysis of the sight centres of the cortex. These include *urœmic amaurosis*, usually transient, in which as a rule the pupillary light reflex is conserved. Some writers, however, ascribe it to a disease of the optic nerve (œdema). There may possibly be both a central and a peripheral form. Transient blindness has also been observed in *lead poisoning*. The blindness due to quinine is also of this kind, but when it is of long duration atrophic changes may develop in the discs. I have also seen a case (along with Seeligsohn [2]) in which the quinine amaurosis was accompanied by reflex immobility of the pupils. It is still doubtful how we should explain the visual disorders which sometimes follow great loss of blood. These sometimes consist in symptoms of a retrobulbar neuritis. Ziegler [3] regards the parenchymatous degeneration of the optic nerve which he found in one such case as an ischæmic necrosis, but this explanation cannot be generally applied. Scagliosi reports changes in the nerve cells after hæmorrhage in acute anæmia. It is remarkable, in this connection, that Bouveret should have found œdema as the only change in the brain in a case in which aphasia and hemiplegia had developed after severe abdominal hæmorrhage. We have no definite knowledge of the nature of the visual disturbances sometimes present in hæmophilia.

Transient hemianopsia may be caused by disturbances which are functional or which are capable of rapid recovery. Thus hemianopsia may occur in migraine as one of the symptoms of an independent temporary partial amaurosis (Flimmer-skotom). It has further been noted as a transient symptom in uræmia and lead poisoning, and in one case it

[1] Hartmann, *Die Orientierung*, 1902; see also Claparède, *Arch. de Physiol.*, 1903.

[2] See Seeligsohn's paper, *B. kl. W.*, 1907.

[3] "Beitr. zur path. Anat.," etc., ii. The question is carefully studied by Wilbrand-Saenger, Bd. iii. Teil. ii. A comprehensive discussion is also given by Singer, Deutschmanns "Beitr. z. Aug." H. liii. See also Assicot, *R. n.*, 1903, and Schultz-Zehden, *Therap. Monatsh.*, 1907.

followed narcosis. It may also be one of the symptoms of a paralytic attack. The form which occurs in migraine is probably due to vascular spasm. In one case only a neurasthenic patient, who did not suffer from migraine, complained of short attacks of hemianopsia which recurred from time to time. In this case also vasomotor disorders may have played a part.

Apart from these conditions, hemianopsia is usually a permanent symptom, due to hæmorrhage, softening, inflammation, new growths, etc. A very transient hemianopsia, however, immediately following an apoplectic attack has been reported as an indirect focal symptom. In one patient under my care, hemianopsia had been the only symptom for twenty-seven years. In another of my cases it was of congenital origin.

I have seen a number of cases in which homonymous hemianopsia yielded to treatment, *e.g.* evacuation of the pus in a case of otitic brain abscess, the removal of a tumour from the occipital lobe, etc. In fracture of the parietal bone the hemianopsia, which was at first total, became later a quadrant-hemianopsia.

Mind Blindness.—This peculiar condition, first observed in animals by Munk—whose explanation, however, was disputed by Hitzig—has also been found to exist in man. In this condition objects are certainly seen, but they are not mentally apprehended. The patient sees, but he is not able to remember the meaning of what he sees; as a patient of Wilbrand's expressed it, she saw only with her eyes and not with her brain. A knife, a key, and so on is seen, but the object wakens no appropriate idea. Thus the patient can no longer orient himself in well-known rooms and streets, because they seem quite strange to him. The power of visual memory is often affected, *i.e.* the patient can no longer spontaneously reproduce visual impressions; he cannot imagine objects, landscapes, faces, etc. This memory power is, however, quite normal in some cases. Mind blindness must not be confused with visual aphasia (see following chapter), although it may be associated with it. A patient with mind blindness is usually unable to estimate distances correctly.

In most cases mind blindness is combined with homonymous *bilateral hemianopsia* of one side and sometimes also with incomplete hemianopsia on the other.

According to Wilbrand [1] the cause of mind blindness is a lesion of the *visual memory fields*, or of the *association fibres* which connect these with the optic centres of perception. If we assume that the shaded parts *O. W.*, in Fig. 301, represent the optic centre in the two occipital lobes, then there is still another cortical region—probably the convex surface of the occipital lobe, and possibly also portions of the inferior parietal lobe—which stores up memory pictures for objects seen during life. We shall call this region, as Wilbrand does the *O. E.*, the *visual memory* field; anything perceived in *O. W.* creates a mental idea only if it awakens corresponding memory images in *O. E.* The visual image is only elaborated in *O. E.* If therefore *O. E.* be destroyed, or if the conduction tracts between *O. W.* and *O. E.* be interrupted (Sachs and Vialet describe fibre bundles which possibly represent such tracts), the patient sees without recognition. If *O. W.* be destroyed on both sides, the patient is actually blind—cortically blind. If *O. W.* be destroyed on one side and *O. E.*

[1] "Seelenblindheit als Herderscheinung," Wiesbaden, 1887; *Z. f. N.*, ii

or the tract *f* on the other, there is unilateral hemianopsia and mind blindness.

This view of mind blindness is opposed to that of Lissauer (*A. f. P.*, xxi.), according to whom the cortical field of the occipital lobe is to some extent merely the retinal field of the cortex. The conception of space and the recognition of objects are only possible because the retinal field of the cortex stands in associative connection with other sensory centres, and especially with the centres for the eye movements: recognition is made possible only by the common action of these various centres. Since we palpate objects with our eyes, as it were, this combination of retinal sensations and of sensations of bulbo-muscular innervation are necessary for a mental sensory perception of objects. But in order to recognise an object the associative connection of the optic centre with the other sensory centres must be intact. Lissauer, therefore, divides mind blindness into two forms: 1. an apperceptive, in which the production of sensory perception is impaired; 2. an associative (transcortical), in which, on account of affection of the association tracts, the connection between the retinal field of the occipital lobe and the centre of another, or with that of the other sensory spheres is interrupted. Claparède (*L'Année psychol.*, 1900), who has collected all the available material, seems to agree with Lissauer. Bernheim regards interruption of the association tracts, which mainly lie in the lateral limiting area of the centre for vision, as the cause of mind blindness. Erbslöh (*M. f. P.*, xii.) has described an interesting case, which he explains in accordance with Lissauer's view.

On the other hand Niessl-Mayendorf (*Z. f. N.*, xxix., and *A. f. P.*, xxxix.) attributes mind blindness to a bilateral lesion of the macular bundle.

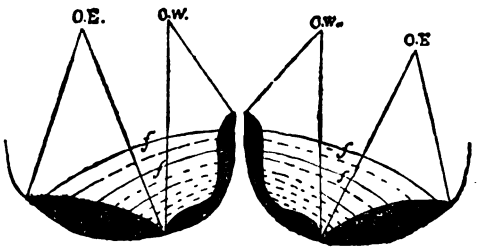

FIG. 301.

Mind blindness is, therefore, usually due to *bilateral* diseases of the *occipital brain*. Bilateral disease of the occipital lobe was found in twelve cases out of twenty which were examined post-mortem. In Liepmann's pure and uncomplicated case also, there were bilateral foci in the occipital lobes, which had left the region of the calcarine fissure intact. It is only in very rare cases of unilateral lesion of the occipital lobe that mind blindness has been observed. In such cases there is usually a new growth which has also affected the occipital lobe of the other side.

Mills (*Med. News*, 1904) has considered the subject in the light of his own experience and that of others. See also the digest by Becke (*Z. f. Aug.*, xi.) and Lenz (*Inaug. Diss.*, Breslau, 1905).

Mind blindness may be recovered from if it is due to the effect of a distant lesion or to lesions which can be recovered from (traumatic encephalitis, gummata, etc.). It may be a passing symptom in paralytic dementia. It may possibly also be caused by inhibition, as in the case reported by Vleuten (*N. C.*, 1904). As a rule it is incurable. It may be simulated by a condition of marked weakness of vision associated with achromatopsia (König-Siemerling [1]).

[1] *A. f. P.*, xxi.

Affections of Speech

For the literature on this question consult Monakow, "Gehirnpathologie," 2nd edition, in Nothnagel's "Handbuch," ix.; also his papers in the "Ergebnissen der Physiologie," the last of which was published in 1907. Moutier's recent work, "L'Aphasie de Broca," Paris, 1908, also gives numerous references to the literature.

The two main forms of speech affection, *anarthria* and *aphasia*, have already been briefly described in the general part. As regards the former, which is identical with affection of the power of forming sounds, we would here emphasise the fact that it is caused by bilateral paralysis or paresis of the muscles of articulation, *i.e.* the muscles of the lips, tongue, palate, larnyx, and respiration. Bilateral lesion of the centres, nuclei, and nerve tracts which supply these muscles is as a rule produced only by lesions at the sites where these tracts lie close together, *i.e.* in the pons and medulla oblongata. In the cerebrum they lie so far apart that persistent and severe affections of articulation can hardly be produced unless by multiple foci in both hemispheres. Unilateral lesions of the facial and hypoglossal centres and of the nerve tracts arising from them have little effect upon the formation of sounds, and any effect that may be so produced is usually transient. There are persons, however, in whom one hemisphere apparently takes the chief part in the innervation of the speech muscles.

The typical speech affection in diseases of the cerebrum is *aphasia*, *i.e.* loss of, or interference with, the power of expressing ideas by words and written signs and of comprehending what is spoken and written in a way that calls up the corresponding idea. That is the definition of aphasia in the widest sense of the word. It may be analysed into a number of component parts, some of which have acquired special names. Thus we are accustomed to speak of the loss of the power of reading, in so far as it is not due to any affection of the sight, as *alexia*; of the loss of the power of writing, independent of any interference with the motility, sensibility, or co-ordination of the arm, as *agraphia*. The most important form, to which the term aphasia was from the first applied, is the loss of speech. Let us try to analyse this more closely. A healthy person has a stroke. On regaining consciousness he finds that he has lost the power of expressing his ideas in words. Although the lips, tongue, etc., can be moved, or are paralysed on the right side only, although sounds can usually be formed, and although we can see from the gestures and from the whole condition that ideas can be normally formed, the power of translating these ideas into words is lost or seriously impaired. The patient can generally say single words, such as "yes" or "no," or even pronounce a senseless repetition of syllables, such as bibi, tatata, etc., but these represent his whole vocabulary. Occasionally this remnant of speech consists of a word or a short sentence spoken immediately before the onset of the paralysis, and now constantly repeated in a stereotyped manner. Whilst *anarthria* is due to paralysis of all the muscles of articulation, with which it is always associated, and dysarthria represents a lesser degree of this affection, the patient being able to form every word, but pronouncing it indistinctly on account of defective articulation (his speech being nasal or slurring), in aphasia it is the *central* process of word formation—the translation of ideas into words—which is partially or completely injured. If we put a question

to an aphasic, he makes a gesture of embarrassment, points to his mouth, and indicates that he cannot speak, or utters the words or sounds which he has retained. One of my patients expressed all his thoughts and ideas by the words "thanks" and "nose." Such patients usually endeavour to make themselves understood by gestures, but occasionally the capacity for expression by means of the facial movements has also suffered (*amimia* or *asymbolia*, see p. 681).

There are cases in which the trouble is due merely to impairment of the power to *produce words*. This has been classified as the *motor* or *ataxic* form of aphasia. It is known as ataxic because the patient sometimes attempts to produce words, introducing wrong sounds and syllables and doubling, displacing, or changing others. It would be better, however, to drop this vague term altogether. The first great discovery in cerebral localisation is associated with this motor form of aphasia, when Broca showed, in the year 1861, that this symptom was produced by a lesion of the posterior part of the third left frontal convolution. Complete motor aphasia is almost always associated with symptoms of paralysis in the right side of the body, but I have seen three cases of this kind in which there was no paralysis although the dumbness was absolute.

In other cases the motor aphasia is associated with another serious symptom: the patient hears, but without understanding what he hears, or grasping its meaning; it is as if he were listening to a foreign language which is quite unknown to him, or only a few words of which he can recognise. We may inquire his name, his age; ask him to put out his hand, etc., but he reacts like a foreigner to whom our language is unknown, or who understands some words and misinterprets the others. This has naturally an extremely depressing and confusing effect upon the patient, and greatly disturbs his mental processes.

This *affection of the perception*, which may be an isolated symptom, is termed *sensory aphasia* (Wernicke) or *word-deafness* (Kussmaul). When it is present alone, the patient is able to speak and to express his ideas, although he cannot understand the speech of others. Close examination, however, reveals the fact that word-production is also affected. Sensory aphasia is due simply to the loss of memory pictures for the sound of words, of the symbols of word-sounds. Every word which enters through our ears leaves behind it in the brain one of these sound images. We are then able to excite this image at will, to let it sound within us, and it is this sound-image which we use when we endeavour to recollect a word. Most people have become independent of calling up these word images in mechanical speech, but not in recollecting or searching for a word. Thus it is that sensory aphasia, the loss of sound-images, carries with it a more or less grave affection of the speech. The patient usually pronounces a number of words or whole sentences without any interruption, especially when speaking of ordinary matters. As soon, however, as it becomes necessary for him to recollect a word, his inability to produce the word-sound becomes evident; he gropes for it and says another word which has somewhat the same sound or sense. On this account he is much more successful in spontaneous speech than in conversation, for should a question or a request cause him to become conscious of his trouble, he no longer speaks mechanically, but becomes confused in speech. The word which reaches his centre

for sound formation rouses it to a certain extent, and as it acts in an abnormal manner its activity has a confusing effect upon spontaneous speech.

A patient suffering from sensory aphasia said to me: "I slept well last night, doctor, I feel exceedingly well, and I hope soon to have quite recovered." After a time I asked: "How are you, Mrs A.?" "Oh, doctor, I am just threely . . . blad, so blad . . . not bad . . . I am again better bad," etc. She had thus comprehended the sense of this question, for which she was prepared, and which was often addressed to her. But the affected centre for sound formation was excited and produced wrong word-sounds, which involved spontaneous speech. In many cases those suffering from sensory aphasia can only speak jargon, like some lately formed language. One of my patients, for instance, said: "Es ja wie wett mam mem hatt miss gern." At the same time there may be an irresistible impulse to talk. It is possible that in such cases there is a lesion of the centre for the formation of sounds which has not only a paralysing, but also a stimulating effect, and therefore leads to the production of meaningless sounds. A. Pick (*W. kl. W.*, 1904) has substituted for this explanation of mine the theory that the sensory speech centre exercises a kind of inhibiting influence upon the motor centre, so that its suppression gives rise to logorrhœa. This view is also advocated by Touche and Freund (*N. C.*, 1904), but I cannot accept it.

Bastian's view, that destruction of the auditory speech centre causes complete loss of speech, does not accord with my experience; in any case it applies only to the first stage of acute affections, in which the effect of the lesion extends beyond the limits of the destroyed area, and the loss of so important a centre affects the whole speech region. Mott and Liepmann have each lately published a case which seemed to show that sudden suppression of the sensory speech centre or of both temporal lobes may cause dumbness, because impulses no longer flow to the motor speech centre, etc.

The interpolation of wrong words, or changing of words, is termed *paraphasia*. This is associated as a rule with sensory aphasia. Motor paraphasia applies to letters, sensory paraphasia to words (Bonhöffer).

The term *amnesic aphasia* has been applied to impairment of the power to call up sound images in the mind and to remember words, when this is the only symptom, and is not associated with any change in the comprehension of words. It forms a variety of the sensory form, and is due to the fact that the sensory speech centre cannot be put into action by other centres, especially by the higher psychical centres, whilst it still responds to stimulation by way of the auditory nerves.

[1] Bischoff and others would include this with the symptoms of partial motor aphasia, or would distinguish between a motor and a sensory form. Heilbronner has recently refused to admit the motor form. See his "Studie über Agrammatismus," *A. f. P.*, Bd. xli.

Quensel and A. Pick (*M. f. P.*, xvi.) thinks that amnesic aphasia is due to a lesion of the second and third temporal convolution.

The term amnesic aphasia has also been limited to cases in which the amnesia for words is due to a general loss of memory (Lichtheim). I must adhere, however, with Eisenlohr, to the definition of amnesic aphasia as described above. Pitres, Trénel, and Hallipré (*Nouv. Icon.*, 1905) have also accepted this view. See also K. Goldstein (*A. f. P.*, Bd. xli.).

The differentiation of these two main forms—motor and sensory aphasia—forms the substance of the whole theory. The localisation of the centre for the formation of sound images in the first convolution of the temporal lobe (and in an adjacent strip of the second) is a positive scientific discovery, which we owe to the genius of Wernicke. He showed that diseases of this region produce sensory aphasia.

The question whether and to what degree the centre for word-sounds coincides with the cortical centre for hearing is still undetermined. As in some cases of word deafness careful

examination fails to reveal any defect of hearing, one is compelled to think that Wernicke's centre for word-sounds is distinct from the hearing centre (Ziehen).

Bezold teaches that of the whole scale of tones it is only necessary to be able to hear the notes comprised between b′ and g‴ in order to comprehend spoken speech. According to Liepmann about two octaves are sufficient for the comprehension of speech, whilst the human capacity of hearing comprises eight octaves. As Wernicke shows, therefore, only some fourth or fifth part of the projection fibres of the auditory nerve radiate into the sensory speech centre. On this question consult also Bonvicini (*Jahrb. f. P.*, xxvi.).

The objections which Marie [1] has lately raised to the prevailing view as to aphasia are directed mainly against Broca's centre and Broca's aphasia. Marie denies the existence of the motor speech centre in the third frontal convolution. It is, according to Marie, only a lesion of Wernicke's site in the left temporal lobe that produces aphasia, the character of which depends upon the intensity and extent of the disease. Involvement of the lenticular nucleus causes anarthria as well as aphasia, the result of this combination being the so-called Broca's aphasia. Moreover, Wernicke's region would thus be not a sensory speech centre, but an area destruction of which would cause a general diminution of intelligence. He also includes in this area parts of the parietal lobe. I regard this theory, as well as some later views of Marie's—on the condition of the intelligence in aphasia—as a complete failure, and Dejerine,[2] Liepmann,[3] and Heilbronner have expressed a similar opinion, whilst Monakow takes up an intermediate position.

Motor aphasia is frequently associated with *agraphia*, even in some cases in which the right hand in not paralysed. An attempt has been made to explain these facts by supposing that writing is preceded by "internal speech," and that therefore the inability to produce words must result in the loss of written speech. Agraphia is, however, by no means a necessary accessory of motor aphasia, and it is certain that, in many persons, written speech is independent of spoken speech.

I have treated a painter, who as the result of an apoplectic stroke had for seventeen years been completely aphasic and could only say "yes" and "no," whilst he could describe the whole course of his illness in writing and painted very well. Ogle, Bonti, Bastian, and Idelsohn (from my policlinic) have published similar cases.

One of my patients developed a complete motor aphasia after an injury to his head; he could only say the word "Anna," and could not repeat what was said to him, but he could write words to dictation. He could also read correctly all the chief words for nouns, but not verbs, conjunctions, etc.

Bastian altogether denies that a lesion of Broca's centre causes agraphia. The theory that the loss of the ideas of movements for writing is the cause of this agraphia is not entirely applicable, as those affected cannot compose words from letters placed before them (Mirrallié).

Agraphia is more frequently associated with sensory aphasia. The explanation has been given that in many (apparently in most) men the sound picture must present itself before the written symbol is awakened, and the impulse thus given for the movements necessary for the production of written signs. No special centre for these ideas of writing movements has been localised in the second left frontal

[1] *Semaine méd.*, 1906; *Bull. et Mém. de la Soc. méd. de Paris*, 1907; *Rev. de la Philos.*, 1907. Also Moutier, "L'Aphasie de Broca," Paris, 1908.

[2] *Presse méd.*, 1906.

[3] *Journ. f. Psychiat.*, ix.

convolution (Exner, Pitres, Ladame, Bastian, Sciamanna, Gordinier); these take place rather in the motor centres, and indeed as a rule in the motor centre for the right hand. This must be directly connected with the visual centre by a nerve path, as we are able to copy what we do not understand and apprehend. Dejerine thinks that the condition of the capacity to write depends entirely upon the inner speech, affection or loss of the latter being the cause of agraphia.

Wernicke (*M. f. P.*, xiii., and "Deutsche Klinik," etc., vi.), to whom we owe the idea of *verbal and literal* agraphia (*i.e.* agraphia for words and letters), thinks that the purely literal form is due to the fact that the visual centre of the cortex, with which the memory images of written signs are connected, is still capable of function, but that its connection with the motor centres which give rise to the movements of writing is destroyed. He bases his view upon observations by Pitres, Pick, Bastian, and himself. In most of these the agraphia was associated with the right hand only, and was frequently accompanied by affections of the sensibility in the right side of the body. In Wernicke's case the agraphia was bilateral, but not entirely pure. In agreement with Pick, he suggests as the site of the disease an area above the posterior margin of the left lenticular nucleus, low down in the white substance of the inferior parietal lobe, near the posterior central convolution in the fasciculus arcuatus. He disputes Dejerine's theory of unilateral localisation of a centre for letters (see below), as in such a case rightsided hemianopia would be necessarily always connected with alexia. He is obliged to admit, however, that the question cannot at present be definitely decided.

Alexia (word-blindness) usually occurs in combination with sensory aphasia. The memory pictures for written characters are apparently situated in both vision centres. In order to set these to work and to convert them into reading, most people have first to produce the corresponding word-sounds. Thus in combining the letters l, o, v, e into the word love, they sound within us and are only then perceived. Injury of the centre for sound formation must therefore also involve the power of reading. Some people, especially when uneducated, read aloud (speaking softly or even loudly), and in such cases lesion of the motor speech centre will also cause alexia.

Alexia has also been noted in some cases as an isolated symptom. The cause is usually found to be an affection of the *left inferior parietal lobe* (angular gyrus), and the explanation is given that the nerve tracts arising from both vision centres pass through the left inferior parietal lobe in order to reach the centre for sound formation (Wernicke, etc.). According to another (Dejerine, supported by Mirallié, Collins, Hinshelwood, and to a certain extent by Thomas and Bernheim and by Ziehen), the left inferior parietal lobe contains a special centre for the formation of letters. Bastian distinguishes between a parietal and an occipital alexia.

In the former the power of recognising letters is retained (Barnett, Hinshelwood). Observations by Hosch, Brissaud, and others also support the possibility of an origin in the occipital lobe. Niessl-Mayendorf (*A. f. P.*, Bd. xliii.) also differentiates a cortico-occipital from a subcortico-parietal origin of alexia, which may be caused by a deep-seated disease of the first inferior parietal lobe from lesion of the dorsal layer of the optic radiation.

An analysis of the mechanism of speech by means of a diagram will greatly aid in the understanding of all these affections. We must, however, keep in mind the fact that such an analysis is always somewhat artificial, and that a great number of cases cannot be made to conform to any diagram. Wernicke, Lichtheim, Charcot, and others have given us diagrams of the speech processes. Although these are not precisely

similar, they yet show us how the mechanism of speech can be arranged. We shall here represent only the most important.

The centre for the word-sound K (Fig. 302 *a*) is produced by words which pass from the ear through the auditory nerve to its nucleus D, and thence to the cortical centre K, in which they leave memory images. The site for the production of words is marked A. It has its origin in the fact that word sounds stored up in K are reproduced by means of imitation, until a similar sounding word is formed. This process continues in A under the control of K, until eventually the conceptions of word movements become so independent in A that they can be directly produced from the idea, B. Some experience points to the fact that even in spontaneous speech the tract B K A is used. The conduction tract which connects A with K passes apparently through the island of Reil. Special bundles, such as the fasciculus uncinatus and arcuatus, are claimed for this process. The impulses pass from A to the nuclei in the medulla oblongata, reaching perhaps first the cortical centres of the muscles of articulation in the foot of the central convolution which, as Rothmann has again recently maintained, control the movements of the speech muscles. For the sake of simplicity ideas are supposed to be formed in a single centre. This is obviously incorrect. We must consider the formation of ideas, which is a very complex process, as connected with the entire cerebral cortex.

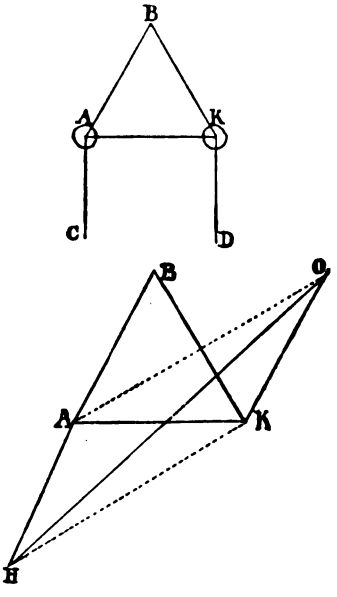

FIG. 302, *a* and *b*.

The conception of a word contains a motor, a visual, and an auditory memory picture. The word "bread," for example, may be resolved into a graphic image, a sound image, a motor memory image for the muscular movements necessary for saying the word, and another for the hand movements required to write it.[1]

The objective idea of bread is composed of a visual image—we see the bread before our mental eyes—of a memory picture of the sense of taste, the sense of touch (arising from handling and eating the bread), etc., and of the memory pictures of the word already described. The visual image and the memory taste image are here most strongly marked, while in the conception of bells, for instance, the sound image is most prominent—the memory of the sound of bells is an essential element.

This is a digression in order to show that the idea of a concrete object is not associated with one centre, but is rather connected with the different sensory centres or consists of a *series of memory images of sensory perceptions*, and may therefore be directly or indirectly aroused by any of these centres. An idea may also be awakened by an allied idea. The sight of a corn-field suggests the idea of bread, etc. The recognition

[1] We need not here discuss the idea of the stereopsychic field described by Storch (*M. f. P.*, xiii.).

of this fact, that memory resolves itself into a sum of partial memories, is of the greatest importance in the diagnosis of morbid conditions. It teaches us to understand that an impairment of speech may be caused by interruption of the tracts which connect the sensory with the speech centres.

It can be seen even from this simple diagram (Fig. 302 *a*) that there may be numerous modifications of motor and senory aphasia according as we believe the disease to be localised in A or K, or in one of the tracts A K, A B, A C, K D. A lèsion of A, for instance, would produce motor aphasia and would render repetition impossible, whilst interruption of the tract A B would indeed remove the power of spontaneous speech, but would not prevent repetition.

Consideration of written speech leads to an extension of the diagram. From the visual centre (for the sake of simplicity represented as a centre O), which is connected with the exterior world by the optic nerve, tracts must pass to other centres (Fig. 302 *b*). It is probably always in connection with K, and in this way reading is made possible. Reading takes place by O K B, or when aloud by O K B A. In some people there is probably a direct tract from O to A. They translate the visual pictures directly into motor, and only understand what they read when they speak it. The process, therefore, follows the path O A B, perhaps even O A K B. There may even exist a direct connection between O and B. In written signs, which are not usually first translated into sounds, but are independent symbols, such as figures, the changing of the written image into the idea takes place in an indirect manner (Monakow). For the right hand a direct tract must go from O to the motor centre (O H). In making a copy the stimulus follows the tract O H. According to some writers, spontaneous writing presupposes a connection of A with H, and this perhaps exists in a number of persons. Probably the tract B K O H is mostly involved, as following the sound image the visual memory image must first call up the written signs before it can be carried over to the motor centre.

Agraphia may be absolute, the patient being unable to make a single written character, or incomplete. Some retain the power of writing their names; others can produce a few letters or can write a few words. If the power of writing spontaneously or to dictation is suspended, that of copying may still be retained. Paragraphia also occurs. Broadbent and Mills assume a special centre for names within the centre for word production, and a corresponding form of aphasia has been observed in Krause's operation of the Gasserian ganglion, resulting from the compression of the left temporal lobe (Hannesfahr). Dana and Fraenkel (*Journ. Nerv. and Ment. Dis.*, 1904) also describe a case of aphasia in which the only function lost was the capacity to name objects.

We would draw attention to the very interesting but rather hypothetical treatise of Mills (*Amer. Journ. Med. Sc.*, 1904).

The different forms of aphasia are variously named according to the conception of these connections, and examples are given to show that almost all these theoretically constructed forms of speech affections do occur.

After Lichtheim we distinguish between *central* and *conduction* aphasias, or following Wernicke between *cortical*, *subcortical*, and *transcortical* motor or sensory aphasias.

In this way the following sub-forms may be distinguished :—

1. *Cortical motor aphasia* : Site of the affection in Broca's convolution—

Voluntary speech
Repetition
Reading aloud
Voluntary writing
Writing to dictation
} Lost or impaired.

Copying
Comprehension of spoken language
Comprehension of written language
} Retained.

(The latter impaired according to Dejerine.)

2. *Subcortical motor aphasia* or pure motor aphasia, or the pure word dumbness of Dejerine (interruption of A C).

Voluntary speech
Repetition
Reading aloud
} Lost or impaired.

Reading
Writing
Comprehension of spoken language
} Retained.

Here the motor memory of speech, the mental speech as a whole, is retained, and the structure of words from sounds and syllables alone is affected, so that a kind of literal paraphasia is present. We can still see from the fragments of words and the inarticulated sounds, etc., that the mental speech process is retained (Wernicke). This condition therefore to some extent forms a link between aphasia and dysarthria. Dysarthritic affections may also be present, as cases by W. König and others show.

Pitres thinks that foci in the white matter just below the speech centre produce the same form of aphasia as cortical foci, whilst a deeply situated lesion, especially if in the left internal capsule, gives rise to this form of speech affection. According to Frankel and Onuf, this form of aphasia is due to interruption of the association bundle which is supposed by them, as well as by Pick and others, to pass from Broca's centre to the articulation centre in the foot of the central convolution. This view is also accepted by Rothmann. Ladame found that the symptoms attributed to the subcortical form occurred in cortical disease, and he therefore does not think that this differentiation is practicable. Bernheim infers from his own careful investigations that the lesion is not always purely subcortical, and that it may involve the cortex also to some extent.

3. *Transcortical motor aphasia* (interruption of A B).

Voluntary speech
„ writing
} Lost or impaired.

Repetition
Reading aloud
Copying
Writing to dictation
Comprehension of spoken and written language
} Retained.

Freund, and also Dejerine, have attributed this form of aphasia to a partial lesion of Broca's area ; this is opposed by Heilbronner (*A. f. P.*, Bd. xxxiv.), who adheres to the transcortical character of the affection. Monakow, Sachs, Bernheim, Bonhoeffer (*Mitt. aus Grenzgeb.*, x.) and Niessl-Mayendorf (*Jahrb. f. P.*, Bd. xxviii.) are opposed to the view of a special transcortical form ; Rothmann, on the other hand, relying on the result of an autopsy, corroborates the existence of transcortical motor aphasia, which he names Lichtheim's form (*N. C.*, 1905 ; *Z. f. k. M.*, Bd. lx.).

4. *Cortical sensory aphasia* (site in K).

Voluntary speech conserved, but verbal paraphasia.
Copying retained.

Repetition Reading aloud Voluntary writing Writing to dictation Comprehension of spoken speech Comprehension of written speech	Lost or impaired.

5. *Subcortical sensory aphasia* (Wernicke's pure word-deafness, Dejerine's pure sensory aphasia).

Voluntary speech ,, writing Copying Comprehension of written speech	Retained.
Comprehension of spoken speech Repetition Writing to dictation	Lost or impaired.

Lichtheim, Bastian, and Sachs attribute this affection to the shutting off of the sensory speech centre from the auditory stimuli which reach it from the periphery.

Freund ("Labyrinthtaubheit und Sprachtaubheit," Wiesbaden, 1895) points out that there is also a word-deafness due to bilateral labyrinthine disease in which sounds are still perceived. This condition may simulate a speech-deafness, and may specially give rise to confusion with the subcortical sensory form. Wernicke has not admitted this fact.

A. Pick and also Dejerine and Sérieux (*Rev. de Psychiat.*, 1898), Veraguth (*Z. f. N.*, xvii.), Strohmayer, and others concluded from their observations that this form of sensory aphasia may be caused by a bilateral disease of the temporal lobes, *i.e.* by a bilateral lesion of the hearing centre, and thus represents an incompletely developed cortical deafness. Wernicke disputed this and rightly showed that a unilateral or bilateral affection of the sensory speech centre causes word-deafness, but that the power of hearing is retained, and that the same is true of interruption of the corresponding subcortical nerve tracts. Then Liepmann (*M. f. P.*, xi.) succeeded in proving that this form of aphasia may be entirely due to a subcortical affection of the left temporal lobe. Dejerine has admitted this. A similar case was reported by Gehuchten and Goris (*Névraxe*, 1901). In order to prove that we are dealing with something different from a form of central deafness, we must carefully test the hearing with Bezold-Edelmann's continuous tone series, taking into consideration Bezold's data, quoted above, as to the range of sounds used in speech, a procedure which Bonvicini has recently followed out (*Jahrb. f. P.*, Bd. xxviii.). He succeeded in demonstrating the absence of any gap in the scale, and thus showed the presence of pure word-deafness. In spite of this, however, much remains still obscure.

In the form caused by bilateral foci, the patient pays no attention to noises (Anton, Bischoff, Pick: see however Redlich's objection, *N. C.*, 1907).

6. *Transcortical sensory aphasia* (interruption in B K).

Understanding of speech or of the sense of words Understanding of writing	Lost or impaired.
Voluntary speech conserved, but paraphasia present.	
Repetition Reading aloud	Retained but without any comprehension.
Copying Writing to dictation	Retained, but without any comprehension.
Voluntary writing conserved, but paragraphia present.	

A symptom which is sometimes present in this form is *echolalia* (Lichtheim, Spamer). Others, however, such as Barr, attribute it to transcortical motor aphasia. Pick regards it as being due to a lesion of the temporal lobe, the inhibitory organ of speech. In our experience it occurs specially in dementia.

Pick and Bastian are inclined to attribute this form of aphasia to a partial lesion of the sensory speech centre. In any case, we are least acquainted with the nature and origin of this form, although if we take a wider conception than Lichtheim, it includes amnesic aphasia, and especially visual aphasia, which will later be more fully described, conditions which are due to the

fact that the centre for sound formation is cut off from its connections with the sensory centres, especially with the visual centres.

7. *Conduction aphasia* (interruption in A K).

Comprehension of spoken speech } Retained.
Comprehension of written speech }
Voluntary speech } Retained, but paraphasia present.
Reading aloud }
Voluntary writing } Retained, but paragraphia present.
Writing to dictation }
Repetition more or less severely impaired.

Wernicke (*Deutsche Klinik*, vi., 1903) stated subsequently that this doctrine of conduction aphasia was still indefinite and threw doubt upon the relation between paraphasia and this form. Ziehen and Kleist have made more recent contributions to this question (*M. f. P.*, xvii.).

Monakow does not believe in a conduction aphasia and points out that there can hardly be purely cortical diseases in the *anatomical* sense; he also states that the anatomical basis of so-called transcortical aphasia is still quite undetermined. Of late his opposition to this view has become still more decided, and he explains the corresponding symptoms by "diaschisis." Compare H. Sachs (*Grenzfragen a. d. Nerv. und Seel.*, xxvi.).

In attempting to localise conduction aphasia we would suspect chiefly a lesion of the island of Reil, *i.e.* of the association tracts within it, but Pershing (*Journ. Nerv. and Ment. Dis.*, 1898) and Niessl-Mayendorf (*A. f. P.*, Bd. xliii.) and others oppose this view.

One point in particular at first received little consideration in the drawing up of these diagrams, namely, that there are great *individual* differences. In the speech of one man the visual memory images are of the greatest importance, in that of another (of the majority) the auditory—he speaks, reads, and writes by means of sound images—and in that of a third the motor. The effect of the disease, which suppresses one of the image-centres, will differ according as this or that memory prevails; in one case it will cause a hardly perceptible disturbance, in another a very severe affection which involves all the factors of speech. We should further remember that we have not always to deal with a complete suppression, but often with a merely partial lesion of an area. These cases, which form the majority, cannot be shown in the diagram. It is evident, as has been stated by Bastian[1] and recently by Bonhöffer and Heilbronner, that diseases which do not completely inhibit the activity of the speech centre, mainly impair its spontaneous excitement, whilst associative stimulation, coming from a sensory centre, often brings it into action. Thus the patient cannot spontaneously find the word "watch," but he says it if the object is placed before him or held to his ear, or he repeats the word after it has been said to him. The explanation is that in lesions of the temporal lobes the understanding of words is very often less affected than spontaneous speech, and that paraphasia or amnesic aphasia mask the word-deafness or precede it. The fact should specially be noted that emotional excitement forms a powerful stimulus for the speech apparatus and may excite it to the production of words and sentences even when spontaneous speech is otherwise suspended. Thus some patients who under ordinary circumstances could not pronounce a word could when in a passion utter an oath.

A Jewish woman, from whom F. Krause had, on my diagnosis, extirpated a tumour compressing the left temporal lobe and the island of Reil, remained for a long time completely aphasic, until one day, when her improvement was being pointed out, she suddenly said "Nebbich."

[1] "Über Aphasie und andere Sprachstörungen." German by Urstein, Leipzig, 1902.

Another symptom which may be mentioned here is that aphasics can sometimes sing and can even find words in singing which they cannot speak.

In a paper published in *Charité-Annalen*, xiii., I have shown that aphasics who could not comprehend written or printed matter nor read them even mechanically, could at once find the right melody for the text of a song shown to them without the musical score. On the other hand there may be complete or partial loss of the memory for music. Edgren, Probst, Larionowa, and others have sought for the localisation of this affection. Probst thinks that the anterior part of the first left temporal convolution is the sensory music centre, whilst motor amusia occurs in focal disease, sometimes of the right (Mann's case), sometimes of the left second frontal convolution. The cases which would point to the existence of a " sensory amusia " without corresponding speech affection, do not appear to me to be very convincing. Interesting contributions have recently been made to this question by Würtzen (*Z. f. N.*, xxiv.), Marinesco (*Sem. méd.*, 1905), Pick (*M. f. P.*, xviii.), F. Alt (" Über Melodientaubheit," etc., Wien, 1906), who describes specially cases of incorrect musical hearing (diplacusis dysharmonica), Auerbach (*du Bois. A.*, 1906), Ingegnieros (*Nouv. Icon.*, xix.), and others.

Aphasia may be due to the inability to retain an image in the memory long enough for it to be transferred to the speech centre. A patient of Grashey's (*A. f. P.*, 1885), for instance, could not read if the letters of a word were only visible to him one after another; he forgot one in reading the next. Gudden, junior (*N. C.*, 1900), describes a similar case. Strümpell also reports a form of aphasia due to disturbance of association.

Finally, in considering the importance of the symptoms, we must remember another factor, namely, the confusion arising from the evoking of false motor images of sound, writing, and words. This confusion arising from the mental processes in speech becomes specially evident if the patient tries to find the names for different objects one after the other. After he has found the word mother, he will call a flower flother. A patient hears the word vakat, and at once immediately gives the word for eye as Ungakat. This clinging to a word once used (perseveration) is a common symptom.

Some writers have been led, by their knowledge of the deficiencies of diagrammatic representations, to formulate a new conception of the nature of the speech affection, which entirely departs from the idea of the speech centres in the old sense of the word, and which regards these merely as the stations at which the different association tracts between the cortical fields of the sensory and motor centres pass through a nodal point, so that lesions of this point interrupt several such tracts. We are, however, absolutely justified in adhering to the old conception of speech centres. We must remember, however, that these are connected in manifold ways with each other and with the central sensory spheres, so that injury of one involves to a certain extent the function of the others, and that individual factors play an outstanding part in speech processes.

Monakow (*N. C.*, 1906) has recently expressed similar views, except that he goes much further and greatly limits the importance of localised speech centres by assuming that in both hemispheres (though specially in the left) cortical territories, extending far beyond their margins, participate in the act of speech. The aphasic symptoms caused by localised lesions of the speech centres are largely due to diaschisis, and it is only the disturbances which persist for a long time and which somewhat correspond to the picture of a partial motor or sensory aphasia which should be regarded as true focal symptoms, etc. The nature of the lesion has also an important influence on the character of the aphasia.

With this limitation we may adhere to the main forms of aphasia and differentiate them as follows :—

1. *Motor aphasia, i.e.* more or less complete loss of speech, the understanding of speech being conserved or but slightly impaired. As a rule,

only a few fragments of words are retained, but the dumbness may be absolute. Writing, reading, and repetition are lost or retained, according to the individual participation of the central processes controlling the reading and writing, and to the special localisation of the process, etc. The construction of sentences is always impaired. In right-handed persons the lesion lies in the posterior part of the *third left frontal convolution*, or in the white substance immediately beneath it. Bernheim and Touche's theory that the motor speech centre extends beyond Broca's area into the second frontal convolution and the island of Reil has no sufficient foundation. In left-handed persons the speech centres lie as a rule in the corresponding areas of the right hemisphere.

A few cases are described (Bastian and Dickinson) in which left-handed persons were affected by aphasia and right hemiplegia as the result of a focal affection of the left hemispheres. It is also a remarkable fact that rare cases occur in which even in right-handed people, in spite of destruction of Broca's convolution, aphasia is absent or very transient (Schreiber, Mingazzini, Collins, Bramwell, Ballet and Armand, and Delille). I myself have seen a tumour which occupied the whole of the third left frontal convolution; the patient was a child. Particular weight has been lately laid upon the occurrence of such cases by Marie, Bernheim, Souques, and Monakow; we meet the same thing, however, in all regions of the doctrine of localisation, because there is here no fixed law and the individual factor plays an important part. We have reason to believe that originally the speech centres were situated in both sides, and that later, under normal conditions, *i.e.* in right-handed people, they became more and more limited to the left region of the cortex, whilst in left-handed people they occupied the right side. It may exceptionally happen, as Bramwell thinks, that the speech centres may be lodged in the right hemisphere even in a right-handed person, and *vice versâ*. Spiller (*Rev. of N.*, 1906) reports a case of this kind. I have observed another along with Heine (ref. *D. m. W.*, 1903), which could not be interpreted otherwise; an abscess in the occipito-temporal region of the right hemisphere in a right-handed person had given rise to visual aphasia which disappeared with the evacuation of the pus.

Aphasic symptoms have been repeatedly described in cases of tumour of the right frontal and temporal lobes. Joffroy (*B. N.*, 1903) thinks that the patients in such cases were originally left-handed and had become right-handed by training, but other cases contradict this view. Mollard (*Lyon méd.*, 1903) also describes word-deafness in a lesion of the right temporal lobe. Senator (*Charité-Annalen*, 1904) observed aphasia with a left hemiplegia, due to an embolic softening of the right temporal lobe in a right-handed person; Mills and Weisenburg (*Med. News*, 1905) report a similar case. I know two cases of this kind. Consult also E. Weber, "Ursachen und Folgen der Rechtshändigkeit." Halle, 1905; Dinkler, *A. f. P.*, Bd. xlii.; Le Fort, *Thèse de Paris*, 1903.

2. *Sensory aphasia, i.e.* total or incomplete loss of the power of understanding speech, although the power of hearing is conserved. Spontaneous speech is usually possible, especially when mechanical, but there is more or less severe impairment of speech when words have to be recollected, or marked trouble if the patient is accustomed to speak with the aid of sound-images. There is sometimes great loquacity with the production of new, meaningless words. Alexia is very frequent and there may be agraphia. Repetition is conserved or lost. The differences are partly due to the intensity, site, and extent of the lesion, partly to individual differences. In right-handed persons the lesion is situated in the *first convolution of the left temporal lobe*, or in its periphery.

3. *Pure alexia* or *word-blindness*, inability to read although speech and the understanding of speech are conserved. Writing is lost or retained (Dejerine). In the latter case (subcortical or pure alexia) the patient can often read whilst writing, tracing the letters with his finger

and by this feeling of movement recalling them to his consciousness. One of my patients who suffered from visual aphasia with quadrant hemianopsia, recognised the letters when one traced them in his hand. In the cases of pure alexia examined post-mortem, lesions have been found in the left occipital or in the *left inferior parietal lobe*. The real cause is probably the lesion of the association tracts between the centre for vision and the sensory speech centre. In such cases right-sided hemianopsia is very frequent. In left-handed persons foci in the right angular gyrus may produce this symptom (H. Köster, Pick, Touche).

The condition may also occur congenitally, as the result of some congenital lesion of the corresponding centres: Stephenson (*Lancet*, 1904); O. Wernicke (*C. f. Aug.*, 1903), Brissaud (*R. N.*, 1904), Foerster (*N. C.*, 1905).

Pure agraphia is very rare. In most people there is apparently no special centre for the memory of movements necessary for the production of written characters. In some people who write directly with the aid of visual memory images, interruption of the nerve tracts which go from these to the left motor centre produces pure agraphia. I have once seen it in its pure form in tumour of the left superior parietal lobe (compare Wernicke's diagram already given). According to Dejerine the cause is interruption of the tract connecting the left angular gyrus with the motor arm centre. If we assume that writing is preceded by mental speech, then interruption of the tract which goes from the motor speech centre to the motor centre of the right hand would cause pure agraphia. In such a case writing with the left hand or with other parts would still be possible. In most people, however, writing is dependent upon the integrity of the centre for sound formation and the tracts which connect it with the motor centre of the hand.

It is possible that agraphia may also be a form or manifestation of apraxia (see above), as Heilbronner (*M. m. W.*, 1906) has suggested.

Visual aphasia (Freund). This form is not uncommon. In it objects are seen and recognised, but cannot be named, although the patient can speak otherwise and can even find the word when the stimulus is awakened from another sensory sphere. For example, when a watch is placed before him, he cannot recollect the word for it, though he knows it is a watch, but he finds the word if it is held to his ear or placed in his hand. One of my patients could not find the name for a thermometer shown to him, but to the question, "What do we use to measure the temperature?" he at once answered, "A thermometer." Another, when *shown* a handkerchief called it a sponge, then a cloth—remembering that it was used to blow the nose, etc., and therefore recognising the object—but he found the word handkerchief the moment it was put into his hand. The patient is therefore unable to call up the visual memory images for speech. Here the lesions are usually found at the margin of the left occipital and temporal lobes and are so extensive that they interrupt the nerve tracts passing from both occipital lobes to the centre for sound formation. The affection is therefore usually associated with alexia, hemianopsia, and often with sensory aphasia, but in one case I could find no trace of word-deafness. Hemianopsia is not, however, a necessary accompaniment. Bilateral foci in the white substance of both occipital lobes may apparently also cause the condition.

I have seen several cases of this (*Fortschr. d. Med.*, 1895) and have been able to show that visual aphasia (as well as the auditory-visual form in which the patient does not comprehend from spoken speech what he can perceive by means of the tracts connecting the centre for sound formation with the vision centres) is not an unusual symptom of cerebral abscess from ear disease. I have seen a number of such cases within the last few years. In one case in which there had been visual aphasia due to a focus of softening in the occipito-temporal region of the left hemisphere, it was still necessary, after the visual aphasia had passed away, for the patient to look at an object for a certain length of time before he could find the name for it. We may suppose that the path from the right vision centre to the speech centre was the only one available, and that this was the cause of the retardation. On the other hand I found no symptoms of visual aphasia in a case in which an abscess had completely destroyed the left fusiform gyrus and part of the cortex of the occipito-temporal and of the third temporal convolution (*Mitt. aus. d. Grenzgeb.* 1900). Marie and Vaschide (*R. N.*, 1903) have lately shown by experiment that the reaction is retarded in aphasics. We should remember that this speech affection is evident not only in the naming of objects shown to the patient, but also in lesser measure under other conditions, as, for instance, in attempting to name an object placed in his hand, where the visual memory image is awakened and thus aids in the finding of the word. Visual aphasia is often not recognised from the disregard of this factor. Von Merkens (*Z. f. Chir.*, 1901) seems to me to have made this mistake. I regard the objections raised by G. Wolf ("Klin. und krit. Beiträge zur Lehre von den Sprachstörungen," Leipzig, 1904) to the theory of visual aphasia as untenable (see, however, the reference by Liepmann in the *C. f. N.*, 1904).

We may also speak of a *tactile aphasia*, but it is exceedingly rare. Although the tactile sense is conserved, objects cannot be *named* from mere touch, as the tactile sense cannot be conveyed to the speech centre. French writers such as Raymond and Egger (*R. N.*, 1906) have lately returned to this term; they have, however, described not an aphasia, but Wernicke's tactile-blindness (Tastblindheit) which they have erroneously interpreted as aphasia. Dejerine (*R. N.*, 1906) had already pointed this out. Following up a paper by Moeli (*B. k. W.*, 1890) I described a case of *true tactile aphasia* (*B. k. W.*, 1890, p. 396) very briefly, because I hoped to be able to report on the results of post-mortem examination, but unfortunately I could not follow up the observation of the case. The power of naming objects by touch was lost or greatly impaired, although she recognised them and could often gradually find her way from the idea to the word. The right hand was neither paralysed nor anæsthetic.

As regards the symptoms and the forms of aphasia we may also mention the following points. Pure forms are very rare; in the majority of cases we have to deal with *mixed forms*, with an aphasia which causes both loss of speech and of the understanding of speech. This aphasia is usually incomplete. If the motor character of the speech affection is strongly marked, the patient can only utter sounds or words; he cannot form a sentence. If the sensory factor preponderates, he can utter whole sentences, but has difficulty in recalling words; he may understand a simple request, but usually he does not, or understands it imperfectly. *Total aphasia*, complete motor and sensory aphasia, is rare.

Word-deafness disappears more rapidly as a rule than the motor affection.

The capacity for making calculations may or may not be correspondingly impaired in the various forms of aphasia. Some cases seem to show that the memory for figures is partly connected with the right hemisphere. I have treated a man with left hemiplegia and hemianopsia who has, since the onset of the paralysis, had difficulty in counting, because he could no longer imagine the figures in their order.

There was also marked loss of power to understand figures in a case described by Bechterew (*N. C.*, 1906).

The aphasic is frequently able to recollect words which are attached

to his memory in a certain order, such as the days of the week, names of the months; to repeat the Lord's Prayer, although he cannot find the same words singly (from the idea). He cannot, for instance, say what month he was born in, although he knows it when he names it in connection with the names of the months.

So-called *agrammatism* (Pick, *A. f. P.*, Bd. xxviii., and *loc. cit.*), *i.e.* speaking in infinitives, in the style of a telegram, leaving out the small conjunctions or adverbs, seems to occur mainly in association with incomplete motor aphasia (Heilbronner, *A. f. P.*, Bd. xli.).

Dyslexia (Berlin) is the name given to a condition in which the patient can only read a few words or sentences, after which he becomes fatigued and objects to continue. This disturbance has been regarded as a partial alexia. Its localising value is still uncertain.

An affection of writing due to abnormal fatigability is described by Gulbenk (*R. N.*, 1904).

I have of late years had the opportunity of observing several cases of a peculiar form of *precipitate speech* (without any sign of aphasia) in patients who also showed other symptoms of a brain disease. In one case it was merely this trouble which led to my being consulted. I have found no evidence as to the nature and cause of the symptom.

Causes of Aphasia.—Aphasia may be of a *functional*, *toxic* nature, and be caused by an *organic* disease.

Aphasia from fright is the type of the functional form. A violent mental shock may paralyse the speech, not merely for the moment, but may produce a motor aphasia which persists for a considerable time (see hysterical mutism). Raymond describes a sensory form of aphasia from hysterical causes, which entirely corresponds to the type of word-deafness. I am sceptical as regards this form. The function of the speech centre may also undergo *reflex* inhibition, especially in children. Aphasic conditions are seen in childhood which are due to irritation caused by worms and which are cured by anthelmintics. A temporary aphasia has also been found to follow a simple operation in childhood.

The aphasia which sometimes accompanies or precedes an attack of migraine is probably caused by vascular spasm and deficient nutrition of the speech centre. I gather the impression from my own cases that there is, independent of migraine, a form of transient aphasia, which reappears from time to time and is caused by momentary vascular spasm, but this must be taken with reserve on account of its great rarity. In *conditions of exhaustion*, loss of blood and inanition, aphasia may be a temporary symptom. Mental exhaustion, sun-stroke, etc., have also been regarded as causes of acute transitory aphasia (Bastian, Rothmann).[1] It is uncertain whether under these and other conditions it is due to a simple congestion, to an intoxication, or to other factors. In Ehrlich's case the aphasia was caused by overloading the stomach with meat that was apparently bad, and it disappeared after the patient had vomited.

Vleuten describes a transitory aphasia in delirium tremens (*C. f. N.*, 1905).

The symptom is more frequent in the course of acute *infective diseases* (especially typhoid and pneumonia). In pneumonia it usually develops on the second and third days, along with apoplectiform symptoms, is

[1] *B. k. W.*, 1903.

motor in character, and may be associated with right hemiplegia. It lasts a few hours or days. It is also seen in *scarlatina*, usually in the later stages when it is due to nephritis, less often at the onset of the attack. In such cases there is probably an infective or toxic agent. This is certainly so in *urœmic* aphasia, which as a rule is very transient. The aphasia which appears in *typhoid* may have the same character, as in an interesting case of Hahn's in which it commenced with urticaria, or it may be due to embolism or thrombosis (Hawkin). It may also persist for a considerable time (Colbertaldo). It is also described as a result of malaria (Plehn).[1] A transient aphasia has been seen in diabetes (Corneille). I have observed several cases of this kind. I[2] have also seen aphasia lasting for one day in a case of carcinoma, without any perceptible alteration in the brain. Transient aphasia has also been noted in santonin poisoning (Dunoyer), and it may also occur in rare cases at the height of an attack of gout. It may be simulated by stupor, in pyrexial conditions, for instance, and by dementia.

In the majority of cases aphasia is due to *organic changes* in the brain, which affect either the speech centres directly, or the white matter lying immediately befow the cortex. Lesions more deeply situated (*e.g.* in the internal capsule, the central ganglia) do not as a rule cause aphasia ; when they do, it is on account of the pressure they exert upon the speech centres. In such cases the impulses from the speech centre may be conducted into the right hemisphere by other paths, *e.g.* by the corpus callosum, and from there onwards.

Hæmorrhages, and still more often softenings—because these more frequently occur in the cortex—are the principal causes of aphasia. Embolism (or thrombosis) of the *left middle cerebral artery* and its branches is one of the most common causes. Hæmorrhages in the external and internal capsule do not produce persistent aphasia ; they form merely an indirect focal symptom which is usually of short duration. In *paralytic dementia* the aphasia is as a rule only a temporary symptom, constituting or accompanying the paralytic attack, but in some cases a persistent aphasia may occur in the course of the disease, quite apart from the terminal stages.

Abscess is a not unusual cause of sensory aphasia in its various forms (especially the visual), as, originating in the left ear, it most often becomes established in the left temporal lobe. Traumatic, rhinogenic, and metastatic abscesses may also produce motor aphasia.

Tumours in the speech centre or its neighbourhood, and especially syphilitic processes, cause aphasia. *Encephalitis* also, as my cases show, and *tuberculous meningitis* may give rise to loss of the power of speech through involvement of the cortex. It has occasionally been attributed to gonorrhœa and to embolic processes (Pitres, Bruns).

Circumscribed atrophy of the cortex in the left frontal or temporal regions may also produce aphasia, as in the cases of Pick,[3] Liepmann, Stransky,[4] and A. Westphal, in which the senile atrophy was very strongly marked at one of these sites. Sérieux and Bischoff have made similar observations. Veraguth and Mingazzini both report similar conditions, and the latter specially states that the changes to which the aphasia is

[1] *D. m. W.*, 1904.
[2] *Charité-Annalen*, xiii.
[3] " Beitr. z. Path., etc., des Zentralnerv.," 1898, and *M. f. P.*, xvi. ; also *M. f. P.*, xix.
[4] *Jahrb. f. P.*, xxv.

due can sometimes be recognised only by means of microscopic examination.

It is also not uncommonly caused by *injury* to the skull (hæmorrhage, splinters of bone, depression of the bone). In one interesting case in which the bone fragment lay upon the speech centre, aphasia could be produced by pressing upon it (Dörrenberg). In one case where the patient had been trephined, I was able to trace the motor aphasia to the tampon which had been introduced between the bone and the third left frontal convolution. When it was removed the aphasia immediately disappeared. In our time especially, when so many brain operations are carried out, we may include not a few cases under the heading of *artificial aphasia*. Bonhöffer has described interesting observations of this kind.

There is a congenital form of word-blindness (see above) and word-deafness (Foy, *Thèse de Paris*, 1905, etc.), the cause of which is as yet unknown.

In circumscribed cortical lesions aphasia may be the only symptom. Motor aphasia is very often accompanied by *hemiplegia* or by left facio-brachial monoplegia, which is explained by the neighbourhood of the motor centres. A lesion may also extend from the motor speech centre deep into the white matter, and in this way involve the motor tracts.

A focus extending deeply inwards from the first temporal convolution not infrequently involves the posterior peduncle of the internal capsule and thus produces, in addition to the sensory aphasia, hemiplegia and hemianæsthesia, and from affection of the most posterior segment of the internal capsule or the optic radiation, right hemianopsia. It is by no means rare for a focus of softening to destroy simultaneously the first left temporal convolution and the angular gyrus; then word-deafness is accompanied by alexia, and usually by right hemianopsia, etc. Lesions of the left inferior parietal lobe situated deeply above the posterior margin of the lenticular nucleus may also cause a combination of hemianæsthesia and agraphia (Pick, Wernicke).

Course and Prognosis of Aphasia.—Aphasia due to functional disturbances and toxic influences generally has a rapid onset and entirely passes away again. This is also the case with the aphasic disturbances following epileptic or paralytic attacks. If it is an indirect focal symptom and due to the pressure from hæmorrhage, it usually disappears in a few weeks or months. Even if it is caused by direct injury of the speech centres, which are not accessible to treatment, the prognosis is still comparatively favourable as regards children and young people. Complete recovery may take place, apparently as the result of the vicarious intervention of the right hemisphere. That the right hemisphere may assume the function of speech even in later youth is shown by a case under my observation:[1] a patient who had been right-handed from her birth, had been compelled by an injury to her right hand at the age of seventeen to make use of the left; she became left-handed. When at the age of fifty-nine she suffered from a tumour of the right temporal

[1] *A. f. P.*, xxi. Wernicke had pointed to this fact in 1874, and the question has also been handled by Entzian (*Inaug. Diss.*, Jena, 1899) and Freund (*N. C.*, 1904). Monakow (*N. C.*, 1906) relies on his doctrine of diaschisis and does not attribute any great importance to the compensatory action of the right hemisphere.

lobe, she became aphasic. Since then Bramwell and Nonne have published similar cases. After recovery from aphasia there often remains for a considerable time an impairment and slowness of conversational speech, sometimes of a peculiar order, which reminds one of the speech of an infant speaking in infinitives, *agrammatism*, etc. (Steinthal, Pick, Heilbronner, etc.), and also dysarthritic disturbances, even a kind of stuttering (A. Pick, W. König). On the other hand I treated a man who had stuttered from childhood ; as an adult he suffered from aphasia with right hemiplegia, and on his recovery from these he had almost entirely lost his stutter. The prognosis depends as a whole upon the curability of the causal disease. Thus *traumatic* aphasia is often cured by trephining, removal of splinters of bone, evacuation of a subdural hæmorrhage. Syphilitic meningitis above the speech centres, gummatous tumours in this region, which cause aphasia, may be absorbed, and the speech affection may then entirely disappear. Sensory or visual aphasia may be cured by evacuation of abscesses of the left temporal lobe. The aphasia may disappear with the removal of a tumour which affects the speech centres without destroying them, as I have sometimes found. I have seen improvement in a case of tumour of the left temporal lobe after evacuation of small cysts, in hæmorrhagic encephalitis of this region, after evacuation of some of the serous exudation by means of brain puncture. The prognosis is most grave in large foci of softening and inoperable tumours. In the former case the aphasia persists definitely although often not in full severity. The patient learns a few words, sometimes even a considerable number, and can often, after a considerable time, make himself tolerably well understood. But even in cases with a favourable course there is often a permanent loss of power of connected speech. The character of the aphasia to some extent determines the prognosis. The so-called transcortical motor aphasia, for instance, tends to disappear more easily and rapidly than the cortical, etc.

Treatment is directed mainly to the causal disease. In aphasia of traumatic origin we have to consider the removal of fragments of bone, foreign bodies, exudations of blood, the chiselling out of a depressed portion of bone, the evacuation of an abscess, etc. If syphilis is present, antisyphilitic treatment is suitable. Abscesses due to ear disease should be evacuated and operable tumours extirpated.

If we cannot treat the causal disease, some improvement of the speech trouble may often be obtained by suitable *training in speaking and writing*. This must be carried out systematically, as in the education of the deaf and dumb, use being made of the tracts by which sensory impressions can still reach the speech centres. The patient who can still read, should learn to speak by reading, simple objects, such as a table, bread, etc., being shown to him, the description being at the same time written below them.

Writing may be used in a similar way. If this faculty is also lost, the tactile and muscular senses may be of use. Small pasteboard letters are put into the patient's hand, which he should combine into words, or his hand should be guided, and a word should be re-written under this guidance until he has comprehended it. He should also learn to imitate the position of the lips and mouth in the different sounds.

I have devoted much attention to these methods in the Charité Hospital, and have permitted them to be described in a dissertation (Nemann, 1884). Gutzmann (see his contributions to

Pentzoldt-Stinzing's "Handbuch," v.; *B. k. W.*, 1901; *A. f. P.*, Bd. xxviii.; *Z. f. physik. Therapie*, viii., and *B. k. W.*, 1907) and others have since sought to extend these methods, by using a stroboscope to read the words from the mouth, etc. Gutzmann thinks it important that the exercises should from the first include the repetition of sounds and meaningless syllables, so that the patient has to read the speech movements by sight. Dànjou, Féré, Thomas and Roux, Mills (*Journ. Amer. Assoc.*, 1904), Bonge (*Inaug. Dissert.*, under Goldschneider), F. Mohr (*A. f. P.*, Bd. xxxix.), and Franz (*Journ. of Philos.*, 1905), have also studied this question. Dejerine and Thomas have obtained results even in longstanding cases. In young persons, learning to write with the left hand may be of great service. As a rule the physician cannot himself undertake this task, as it requires much time and great patience. The relatives, or better still, a teacher of language, should be responsible for this education. The result is often negatived by the diminution of intelligence due to the brain disease, or by the rapid fatigability of the affected brain. One of my patients first learned the alphabet like a child by means of pictures. He could not pronounce the *e* and *i* when they were written in front of him, but he could find them with the help of an eagle and an idol, although he was always apt to say *eag* instead of *e*, and *id* instead of *i*.

Appendix: Examination of an Aphasic.

The following is the best method: we must first ascertain that the patient can hear before we test his understanding of words. For this, it is necessary to pass from simple requests, such as, "Touch your nose"; "Put out your tongue," to more complicated questions. Very careful investigation is required to recognise slight degrees of word-deafness. We must avoid helping the patient by gestures. Ask him to name objects shown to him, parts of the body, etc. If he cannot find the words for them, we must ascertain whether this is due to visual or motor aphasia. In the former case we can usually find the word by stimulation from another sensory sphere, or from the idea; we should therefore place the object in his hand or ask, if he cannot name a key, for instance, whether he knows the instrument with which we open a door, wind up a watch, etc.

After we have tested his power of naming concrete things, we should examine his capacity for connected speech. We should specially note whether he has to search for words, to consider some of them for a long time, to confuse them, etc.

We should then discover whether he can repeat, read, and write. If he has lost the power of reading aloud, he is perhaps still able to apprehend the sense of written matter. We should therefore write down a simple request. We should also distinguish between the recognition of letters and of words.

In writing, the power of simple copying, of writing to dictation and spontaneously, should be tested.

There is no practical interest in extending the examination to the musical faculty.

We should above all be careful not to fatigue or confuse the patient.

AFFECTIONS OF THE MENINGES

Inflammations of the Dura Mater

(*Pachymeningitis interna hæmorrhagica, Hæmatoma of the Dura Mater, etc.*)

Pachymeningitis externa does not occur as an independent disease. It develops in connection with affections of the bones of the skull, which may be of traumatic origin or dependent on caries, osteomyelitis, syphilis, erysipelas, tumours, and the like. The symptoms of pachymeningitis are thus frequently masked by those of the primary disease. Suppuration spreading extradurally from the ear may give rise to characterisitic features (see chapter on brain abscess). The affection is principally of surgical and otological interest.

Pachymeningitis interna hæmorrhagica and *hæmatoma of the dura mater* are of greater clinical importance.

Pathologically the former is well known; it is only as regards its mode of development that opinions differ. According to Virchow,[1] and Heschl,[2] inflammation of the dura mater is the primary cause, and it gives rise to the formation of a stratified membrane on the inner aspect of the dura. This is very vascular, and hæmorrhages occur from rupture of the vessels. Melnikow-Raswedenhow [3] and Barrat [4] have more recently come to the same conclusion from their observations. Others, such as Huguenin, Spiller, and M'Carthy are of opinion that the hæmorrhage is the primary change, the connective tissue membranes resulting from the organisation of the clot.

The post-mortem examination in slight or early cases shows only slight alterations, *e.g.* a delicate pink or greyish red deposit on the inner surface of the dura mater, in the form of a veil-like, detachable membrane, which is usually studded with punctiform hæmorrhages, speckled here and there with brown and yellow from the deposition of pigment derived from older blood effusions. The dura is slightly distended, most frequently over the parietal lobes, but it may be over the whole hemisphere or even on both sides. The deposit may also be found at the base, especially in the middle and posterior cranial fossæ. The process sometimes reaches such an intensity as to lead to the development of successive layers of firm membrane, which cover the brain like a thick cap, between dura and arachnoid (see Fig. 303), and become adherent to these membranes and here and there also with one another, while recent and old hæmorrhages, which may be as large as a goose's egg or a fist, extend through and between the laminæ. The younger layers are delicate and hyperæmic, the older, solid, hard, and almost bloodless. Occasionally the process leads to new formation of bone. The brain, especially the cortex, is more or less firmly compressed and atrophied. A condition of softening is found in rare cases.

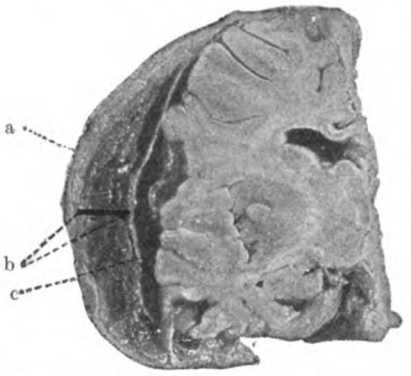

FIG. 303.—Frontal section through the cerebral hemisphere of a child who died of internal hæmorrhagic pachymeningitis. *a* = dura. *b* = organised clot. *c* = newly formed membrane. (After Misch-Finkelnstein.)

Pachymeningitis interna hæmorrhagica is for the most part an accidental affection. The milder grades form an occasional accompaniment of phthisis and chronic heart and kidney conditions. It is specially associated with brain affections of a chronic inflammatory nature, which lead to atrophy of that organ, and is commonly found in general paralysis, chronic hereditary chorea, and senile dementia. It cannot be doubted, however, that it must also be regarded as a primary disease. It occurs

[1] "Verhandl. d. phys. med. Ges.," Würzburg, 1856.
[2] "Kompendium der allg. und spez. Path.," 1855.
[3] *Ziegler's Beitr.*, Bd. xxviii.
[4] *Br.*, 1902. See also Jores, "Verhandl. d. deutschen path. Ges.," 1899; Laurent, "Dissert.," Bonn, 1898; Vleuten, "Dissert.," Bonn, 1898; Fürstner, *A. f. P.*, viii.

relatively frequently in chronic alcoholism. It has been observed also in affections dependent on a hæmorrhagic diathesis, such as pernicious anæmia, leukæmia, scurvy (Magnan, Broer), Barlow's disease, purpura (Lépine, Obernier, Havas), anthrax (Wagner, Curschmann, Ziemke), hæmophilia (Moizard), and in cases with merely a hæmorrhagic diathesis (Berggrün). Syphilis is a rare cause (Beck, Hahn). It develops sometimes as a sequel to acute infective diseases (small-pox, scarlatina, typhoid). Subdural hæmorrhages have also been seen to appear in the course of whooping-cough ; subarachnoid hæmorrhages may be due to a ruptured aneurism or the bursting through of a hæmorrhage from the ventricles. In certain constitutional states (alcoholism, nephritis) convulsive attacks may also give the start to the development of these meningeal hæmorrhages (Luce [1]).

Finally, *injuries to the skull* often pave the way for pachymeningitis hæmorrhagica interna.

I have several times seen in elderly individuals with obstinate constipation, requiring excessive abdominal pressure, the development of features which I ventured to believe were due to pachymeningitis hæmorrhagica. I have, further, observed two cases in which I was compelled to attribute the disease to over-vigorous acts of coitus. In one of the cases there was years afterwards a return of the condition from the same cause.

It would appear from the observations of Herter, Legendre, Göppert [2] and Finkelstein,[3] etc., that the affection occasionally occurs in childhood.

Hæmorrhages, chiefly subdural, resulting from severe labours, may extend over both hemispheres, also on to the tentorium and round the cerebellum (M'Nutt, F. Schultze). We are indebted to Cushing (*New York Med. Journ.*, 1907) and Seitz (*A. f. Gyn.*, Bd. lxxxii., and *M. m. W.*, 1908) for important observations on this question.

There may be no symptoms throughout the disease, especially if we are dealing with slight or accidental changes. In paralytic dementia it is not as a rule revealed by any special symptoms. In a number of cases, indeed, it is masked by the symptoms of a brain affection, which, however, are not sufficiently characterisitic. They entitle us nevertheless to make a probable diagnosis if they appear under conditions in which we know by experience that hæmatomata of the dura mater tend to develop, *i.e.* in alcoholism, after injuries to the skull, in senile dementia (Jahrmärker), etc. It is obvious that the symptoms will vary greatly according to the site and extension of the process. In the majority of cases in which the disease is an entity *per se* the symptoms are as follows : The patient, generally after a *state of excitement* with violent motor unrest, which may correspond to delirium tremens, or after a stage of intense headache, vomiting, and *unilateral epileptic convulsions*, falls into a state of coma which may last for days or even weeks. During this period the *pulse* is usually slowed and sometimes irregular, and respiratory disturbances occur. More rarely the affection sets in with an *apoplectic attack*. During the coma *paralytic symptoms*, for the most part *hemiparesis or monoparesis*, tend to appear ; the latter may gradually develop into complete hemiparesis and may spread to the other side. Bilateral paralytic symptoms are often present from the outset. *Hemiparesis* of the one side may be associated

[1] *Z. f. N.*, xiv. [2] *Jahr. f. Kind.*, 1905. [3] Misch, *Jahrb. f. Kind. N. F.*, Bd. lxii.

with a contracture of the other. Stiff-neck, contracture of the leg, and Kernig's sign are sometimes present. The *sensibility* is not usually affected to any extent, but well-marked hemihypæsthesia sometimes occurs. *Aphasia* has been noted only in some cases. *Unilateral or general convulsions* appear not infrequently, and Luce lays great stress upon their frequency and violent character as favouring the diagnosis. Nevertheless the paralysis and convulsions are often only of fleeting duration. Frequently they take the form, not of typical Jacksonian attacks, but of irregular twitchings, appearing now here, now there, or of automatic choreiform movements, constrained attitudes, or the like, which are provoked by the compression. It should be noted that *choked disc* has been found more than once (due to effusion of blood into the optic sheath), and usually only on the side opposite to the paralysis.

Choked disc is found in pachymeningitis interna hæmorrhagica of non-traumatic origin (Michel, Wernicke, Manz, Fürstner, and others), but is specially produced by traumatic dural and arachnoid hæmorrhages (Panas, Bergmann, Uhthoff, and others). It depends, therefore, as a rule on considerable hæmorrhages at the base of the skull, usually due to rupture of the middle meningeal artery, with or without fracture of the optic canal.

In the cases complicated with fracture of the base, Fleming found also retinal hæmorrhages, especially on the same side as that of the fracture. I have also personally observed this. On this subject see further Liebrecht (*A. f. Aug.*, Bd. lv., and *Disc. N. C.*, 1906), and for the literature, Uhthoff in Graefe-Saemisch "Handbuch," 2nd Ed., Bd. xi. Kap. xxii.

Conjugate deviation of the head and eyes is present in a number of cases. Nystagmus is sometimes observed. The *pupils* are usually at first contracted, later that on the side of the lesion may be dilated. When coma is absent or is clearing up there is as a rule severe dull *headache*, which may also precede the coma, as well as vertigo, staggering gait, etc. Local tenderness of the skull to percussion is an important, but by no means constant symptom.

Remissions and exacerbations occur very frequently; the coma may from time to time diminish and then deepen again. These recurrences are quite characteristic of a chronic course.

The temperature frequently rises above normal at the very onset, then usually rises higher, and before death may reach a height of 41° C. In chronic remittent cases I have seen rises of temperature from time to time. Subnormal temperatures also occur.

The disease may be rapidly fatal or may run a prolonged course. Complete recovery is rare in severe cases, usual in slight ones. Death usually results in chronic cases, after months or years.

In the comatose stage it is difficult to distinguish the affection from meningitis; nevertheless as a rule the stiffness at the back of the neck is absent or it does not reach such a high degree, and the cranial nerves (with exception of the optic) are practically always spared. Paralysis of the basal cranial nerves only becomes marked in the more rare cases in which the hæmatoma extends to the base. An interesting case of this kind in which opthalmoplegia was the predominating symptom, has been described, *e.g.* by Ziehen.[1] Under these conditions, as in a patient of Nothnagel's, it may also lead to the symptom of rigidity of the neck. Heitz and Lamy also describe cases of this sort, and Achard-Ramond [2] record an instance in which a cerebral hæmorrhage bursting into the meninges simulated an acute meningitis.

[1] Ebstein-Schwalbe, "Handbuch d. prakt. Med.," iv., 1900.

[2] *R. n.*, 1904.

When the onset is apoplectiform the further course is important for diagnosis; the inconstancy of the paralytic symptoms, the intercurrent appearance of convulsions, and the choked disc of this disease make it impossible to exclude with certainty a genuine cerebral hæmorrhage. Nonne [1] and Apelt [2] have shown recently that it may be quite impossible to distinguish them, and that even thrombosis with cerebral softening may give rise to the appearance of a pachymeningitis hæmorrhagica or a hæmatoma of the dura mater.

Jahrmärker succeeded in making a diagnosis from the clinical appearances in cases where the disease occurred in the course of senile dementia (*M. m. W.*, 1907).

Courmont and Cade regard the variety and the rapid alteration in the symptoms as specially valuable in the differential diagnosis from cerebral hæmorrhage. In isolated cases the disease has been confused with general paralysis.

In doubtful cases *lumbar puncture* may enable one to decide (see p. 750). It is specially applicable to the traumatic form of the disease and has often placed the diagnosis on a sure footing. A hæmorrhagic form of cerebro-spinal meningitis also occurs, as is shown, *e.g.* by an observation of Achard-Grenet.[3] Puncture of the skull, according to Neisser's [4] method, is of still greater value in clinching the diagnosis, as is shown by the results of this author and his co-worker Pollack,[5] the observations of Nonne-Apelt and Misch,[6] and as I have personally observed. If a large quantity of blood can be aspirated, brownish, dark brown, blackish, or mixed with serum, then the diagnosis of hæmorrhage is assured. The authors lay stress upon the fact that the blood of this hæmatoma, as a rule, remains long uncoagulated and that in a hæmorrhage which has spread chiefly extradurally it also remains more or less fluid for even a still longer time.

Treatment.—Therapeutic measures are limited practically to careful guarding against injury, application of an icebag, and perhaps bleeding in plethoric persons. Free evacuation of the bowels is also recommended. In one case of a patient in whom I had attributed the sudden onset of cerebral symptoms to pachymeningtis hæmorrhagica, they all disappeared in a few weeks, under the régime of bleeding and diaphoresis, which I had recommended. In severe, and to a certain extent also in merely impending cases, it may be advisable to employ *puncture of the brain* or puncture of the skull. As the experience of Neisser and Pollack shows, one sometimes succeeds in drawing off so much blood in this way that the severe symptoms pass off and recovery follows. In other cases the radical operation must be employed. In some instances—for further consideration, see under Traumatic Hæmatoma—repeated lumbar puncture has led to recovery.

Some writers, *e.g.* Bass (*C. f. Gr.*, 1900), Taillens (*Revue méd de la Suisse*, 1902), Misch, Finkelstein (*Jahr. f. Kind.*, Bd. lxii.), praise the use of subcutaneous injections of gelatine preparations.

Traumatic meningeal hæmorrhages,[7] especially *traumatic hœmatoma* of the dura mater, requires special consideration both as regards sympto-

[1] Ref. *D. m. W.*, 1907.
[2] "Mitt. aus Grenzgeb.," xvi. and xviii.
[3] *R. n.*, 1904.
[4] Neisser-Pollack, "Mitt. aus Grenzgeb.," xiii.
[5] "Mitt. aus Grenzgeb.," xviii.
[6] *Jahr. f. Kind.*, xii.
[7] Literature: Bergmann, "Die Lehre von den Kopfverletzungen," *D. Chir.*, Bd. xxx., 1880; Marchant, "Les épauchments sanguins intracraniens, etc.," Paris, 1881; Krönlein, *Beitr. z. kl. Chir.*,

matology and treatment. Extra- or epidural hæmorrhages result chiefly from rupture of the meningeal arteries or one of their branches. Injuries to the meningeal arteries are by far the most common cause of all the traumatic intracranial hæmorrhages (Marchand, Hewett), and as a matter of fact the middle meningeal artery is almost exclusively involved. Subdural hæmorrhages may arise from laceration of veins in the pia mater or of the veins opening into the superior longitudinal sinus. A large proportion of the hæmorrhages occurring in the child during birth have their origin in this way. Rupture of the sinus itself may also bring about this condition. Hæmorrhages of both varieties may result traumatically without any rupture of continuity in the bones. This has been shown especially by Bergmann and proved by the experiments of Felizet; Ledderhose and Brion have also reported cases of this kind. On the other hand subarachnoid hæmorrhages occur almost exclusively in combination with severer injuries which affect the brain also, and they are therefore not of such special interest.

The site of the hæmorrhage usually corresponds to the site of the injury, but the arterial laceration may through *contre-coup* take place on the opposite side (Wiesmann) or on both sides.

Opinions recently expressed against the theory of *contre-coup* do not appear to be of much value.

The amount of blood effused may reach 200 grm. or more. Krönlein divides the hæmatomata, according to their extent, into diffuse and circumscribed forms, and subdivides the latter into anterior (fronto-temporal), middle (temporo-parietal), which are by far the commonest, and posterior (parieto-occipital).

From the stupor directly due to the injury, which may have all the appearances of cerebral concussion but which may be very slight or altogether absent, the patient wakes up to full consciousness, but only to lose it again after a certain time. In middle meningeal cases the free interval may last from quite a short time up to twenty-four hours; more rarely it may even extend to several days or a week and more (Broer). We owe to Raymond an interesting observation of this kind. In subdural and intermeningeal hæmorrhages the free interval, on an average, lasts longer (Allen Starr), even for weeks or months (cases of H. Fischer, Köhl, and others); it has even been maintained that the first evident signs of injury to the brain may not appear until a year has passed, but reports of this sort must be received with the utmost caution.

When the interval amounts to days or weeks (*late traumatic apoplexy*), one is dealing often with a hæmorrhage, not in the meninges, but in the neighbourhood of the aqueduct of Sylvius and the third and fourth ventricles, the result of a local softening, and therefore of secondary significance.

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xiii., *Z. f. Chir.*, xxiii.; Kocher, *Z. f. Chir.*, 1893; Moritz, *Vierteljahrschr. f. pr. M.*, 1892; Brion, "Die operative Behandl. der intraduralen Blutungen," Dissert., Strassburg, 1896; Broer, "Dissert.," Breslau, 1900; Wiesmann, "Handbuch d. prakt. Chir.," 1900; Kocher, Nothnagel's "Handbuch," ix.; Phelps, "Traumat. Injuries of the Brain," New York, 1897; Ortner, *D. m. W.*, 1897; Raymond, *Presse méd.*, 1903; Bruns, *Beitr. z. klin. Chir.*, Bd. xxxviii.; Gebauer, *W. kl. R.*, 1903; Göppert, *Jahrb. f. Kind.*, 1905; Sänger, *N. C.*, 1905; Herford, "Friedr. Blätter," 1905-6 Yoshikawa, *M. f. P.*, xx. Ergänz; Nonne, *N. C.*, 1906; Apelt, *Mitt. aus d. Grenzgeb.*, xvi.; Nonne, *D. m. W.*, 1907; Pringle, *Scot. Med. and Surg. Journ.*, 1906; Enderlen, *Z. f. Chir.*, Bd. lxxxv.; Cushing, *New York Med. Journ.*, 1907; Ballance, "Some Points in the Surgery of the Brain," etc., London, 1907.

(Duret,[1] Bollinger,[2] Matthes,[3] Stadelmann,[4] Bohne,[5] Gebauer,[6] Langerhans,[7] O. Israel,[8] Rupp,[9] and others; consult the chapter on cerebral hæmorrhage.)

After passing through a stage of delirious excitability, the patient, usually within a few hours, becomes somnolent; the somnolence deepens into coma, the pulse becomes slow and wiry, and the breathing is usually deliberate and stertorous. These symptoms are due to the compression and increase of intracranial pressure. According to Pagenstecher, they only appear when the hæmatoma exceeds 37-40 c.cm. in volume (further details on this point have been collected by Apelt). Choked disc sometimes develops but it may quickly disappear again; this corresponds with Schulter's experimental results. There may be in addition signs of unilateral, or more rarely of general motor irritation, unilateral spasms, unilateral or bilateral contracture, stiffness of the neck, contracture of the legs, or Kernig's sign, and paralytic symptoms usually of an incomplete hemiplegic character. Bilateral hemiplegia sometimes develops. Finally, it is in traumatic hæmatoma that the so-called collateral hemiplegia has most often been recorded (Bergmann, Moullin, Wiesmann, Ledderhose, and others). It has already (p. 686) been pointed out that this assumption rests in most cases on a diagnostic error. A bilateral hæmatoma is occasionally present; it is more extensive on the side of the injury, but on the other side is so situated that it affects the motor cortex. Aphasia is no uncommon feature. Hemianæsthesia and hemianopsia have been less often recorded, but an extensive hæmatoma may, undoubtedly, by compression of the cortex, bring about a well-marked hemianæsthesia (Oppenheim). Fever, even up to 40° C. (104° F.), albuminuria, and glycosuria may be amongst the symptoms. Respiratory disturbances of the type of Cheyne-Stokes breathing occur (Touche, Grisson, Sänger, Oppenheim).

The pupil on the side of the hæmorrhage is often dilated, and choked disc may occur only on this side. It is always very difficult to localise the site of the hæmorrhage so long as the patient remains in a state of coma. Ortner believes that, in doubtful cases, the behaviour of the respiratory muscles gives valuable evidence, since the unilateral paralysis of these, naturally on the side opposite to the injury, may be recognised even during the comatose stage (compare, however, p. 685). In cases of this kind the condition of the plantar and leg (Oppenheim's) reflexes may perhaps also lead to a more certain diagnosis as the extensor type appears especially on the side opposite to that of the hæmorrhage. The coma, moreover, is not always complete, or it may clear up, leaving a certain stupor, the diagnostic value of which has been pointed out by Kocher. Localised tenderness of the skull to percussion and pressure may also afford valuable indications as to the site. In the rare cases in which pachymeningitis hæmorrhagica extends to the base, signs of paralysis of the cranial nerves become prominent. By compression of the basal regions of the brain the effusion may produce special features, such as temporal-lobe symptoms or bulbar paralysis. We owe to Neisser-Pollack a case of this kind in

[1] "Études expér. et clin. sur les traumatismes cérébr.," Paris, 1878.
[2] "Über traumat. Spätapoplexien. Internat. Beitr. z. wissensch. Med. Festschr.," R. Virchow, 1891.
[3] "Volkm. Samml. kl. Vortr.," *N. F.*, 322, 1901.
[4] *D. m. W.*, 1903.
[5] *D. m. W.*, 1903.
[6] *W. kl. R.*, 1903.
[7] "Die traum. Spätapoplexie," Berlin, 1903.
[8] "Vierteljahrschrift f. gerichtl. Med.," 1903.
[9] *Z. f. Heilk.*, 1905.

which the symptoms had their origin in the posterior fossa. In an interesting case of Ruoccos' the basal hæmorrhage was associated with a fracture of the odontoid process.

In hæmorrhages which extend into the subarachnoid space, or into the spinal meninges, corresponding symptoms may appear, *e.g.* Kernig's sign. The tendon reflexes even disappear in such exceptional cases as that recorded by Saenger. In this connection the experimental results of Yoshikawa should be taken into consideration.

If medical aid is not given the patients as a rule die in a comatose condition; but the general symptoms may, on the other hand, disappear, while the local symptoms (hemiplegia, Jacksonian epilepsy, etc.) persist for many years, as I have personally seen and as is shown by numerous reports of cases of Jacksonian epilepsy of traumatic origin which have been operated upon. In this case the organisation of the blood-clot and the chronic inflammation of the surrounding meninges will have produced indurated fibrous plaques, which may enclose foreign bodies, *e.g.* splinters of glass, as I have seen in one case of Bergmann's. Meningeal cysts may also result from a process of this kind.

Other cases occur in which the symptoms are indistinct and vague: the patient complains of transient dizziness, pain in the head, loss of memory; he is slightly stupid, but motility is more or less completely preserved, and it is only on careful examination that objective signs of a cerebral lesion, such as slight hemiparesis, partial aphasia, slowing of the pulse, or such like are detected. Raymond, for instance, describes a case of this sort where the patient came for consultation to the Salpêtrière.

Attempts have been made to decide from the clinical appearances whether the site of the hæmorrhage is inside or outside of the dura (Brion, Allen Starr, Schultze, Gebauer, and others), but the criteria adduced cannot be said to be absolute. It is quite true that in pachymeningitis interna hæmorrhagica the general symptoms of cerebral compression develop as a rule much more slowly, but the same holds good in the main for the non-traumatic form.

The distinction between traumatic hæmatoma and cerebral contusion or cerebral shock may be so difficult that even the most experienced physician may make a wrong diagnosis (Stolper, Kocher, Schultze, and others).

An important point in the differential diagnosis is, that in cerebral shock the symptoms reach their greatest intensity at the very onset, and that there is no free interval, etc. But, with Kocher, Pringle, and others we must exercise the greatest caution in the comparison and diagnostic separation of these traumatic cerebral affections, as they often appear in combination. Quite recently, Nonne and Apelt have specially pointed out the great uncertainty of the diagnosis, and have shown that confusion with cerebral hæmorrhage and even with cerebral softening is possible. In one instance thrombosis of the middle cerebral artery with encephalomalacia simulated the appearances of a traumatic hæmatoma. Special care is required when the patient is an alcoholic subject. According to an observation of L. Müller's, symptoms of a hæmatoma of the dura mater may occur, in cases which show no detectable pathological change.

Subarachnoid serous effusions, which it may be impossible to distinguish from hæmorrhages (Walton, Bright, Godlee, Ballance), sometimes follow injury to the head. Göppert has seen a combination of pachymeningitis hæmorrhagica with hydrocephalus externus in children, and Ballance has made similar observations.

Lumbar puncture has been frequently employed during the last

decennium to establish a diagnosis of traumatic hæmatoma or of meningeal hæmorrhage. One would suppose that the result in a hæmatoma of the dura would be negative, since the subarachnoid space of the spinal cord contains only a sanguineous fluid which can be obtained by puncture when the site of the hæmorrhage is subarachnoidal. Ballance has expressed his opinion to this effect. Quincke reports that in extradural or intermeningeal hæmorrhage without injury to the arachnoid, there will be found at most only a yellow tinting of the fluid by diffusion. But apart from the fact that intermeningeal hæmorrhage is sometimes associated with an epidural hæmatoma, blood coming originally from the latter may by bursting through the dura and arachnoid reach the subarachnoid space. A considerable literature has already grown up around this question.

I would refer to the earlier communications of Braun, Poirier, Nothnagel, Sicard, Netter, Tuffier, Terson, Bard, Sainton-Ferrand, Demoulin, Pavy, and to the more recent ones of Milliet, *Thèse de Paris*, 1902; Mathieu, *Thèse de Paris*, 1902; Bard, *Sem. méd.*, 1903; Widal, *Presse méd.*, 1903; Froin, *Gaz. des hôp.*, 1903; Froin, *Thèse de Paris*, 1904; Froin-Boidin, *Gaz. des hôp.*, 1904; Chauffard-Froin, *R. n.*, 1904; Dupré, *N. C.*, 1904; Potherat, Quénu, *D. m. W.*, 1905; Hérault, *Thèse de Paris*, 1905; Rindfleisch, *A. f. kl. M.*, Bd. lxxxvi.

These observations, made especially by French physicians, give the following results: In cerebral affections following on head-injuries (with or more rarely without fracture) the fluid most frequently contains blood or the elements of the blood. This appearance of blood or the brownish-yellow tinting, not merely in the first fluid drawn off but also in the later samples, is as a rule conclusive (chromodiagnosis). Confusion with blood derived from puncture of a vein is obviated by the fact that this quickly clots, while blood coming from the cerebral or cerebro-spinal meninges loses its capacity for coagulation more and more as it mixes with the cerebro-spinal fluid (Henneberg, Finkelstein, Milliet). In doubtful cases direct observation must be supplemented by microscopic examination.

The blood may disappear from the fluid within a few days, the red colour then giving place to a yellow tinge (Milian, *Gaz. hebd.*, 1902). In a case described by Nothnagel an intermeningeal hæmorrhage coming from a ruptured aneurysm was diagnosed in this way; interesting cases of a similar nature are reported by Widal (*Presse méd.*, 1903), Bauer (*Arch. gén. de Méd.*, 1903), and Ohm (*B. k. W.*, 1906).

It is shown, however, *e.g.* by a report of Roussy's (*R. n.*, 1905), that meningeal hæmorrhages occur without admixture of blood in the cerebro-spinal fluid.

In view of this uncertainty of the results it certainly seems advisable to confirm lumbar puncture by Neisser's skull puncture, particularly as, according to Neisser-Pollack and to Pollack and Nonne, this also makes a topical diagnosis possible. In some cases it also appears to stimulate directly the process of recovery, and to save the patient's life.

Treatment.—If hæmorrhage from the middle meningeal artery is diagnosed, operative treatment, *e.g.* ligature of the bleeding vessel and clearing-out of the accessible clot, is urgently indicated. Palliative treatment by ice-bags, bleeding, etc., is of little value. On the other hand spontaneous recovery has been observed in at most 10 per cent. of the cases (Wiesmann).

Of recent years *surgical* treatment of traumatic hæmatoma of the dura mater has had gratifying results.

We owe communications on this subject to Macewen, Starr, Ceci, Wagner, Krönlein, Hahn, Köhl, Raymond (*Méd. mod.*, 1902, and *Presse méd.*, 1903), Jackson, Grisson-Sänger (*N. C.*, 1903), Taylor-Ballance (*Lancet*, 1903), Nonne-Apelt, Newman (*Lancet*, 1903), Vincent (*Arch. prov. de Chir.*, 1905), Caveillon (*R. n.*, 1905), Peairs, Sommer, Buzzard (*Lancet*, 1906), Nötzel (*D. m. W.*, 1907), Krönlein (*A. f. kl. Chir.*, Bd. lxxxi.), Anschütz, Cushing (*New York Med. Journ.*, 1907).

Raymond's case is also of interest, inasmuch as the operation on the hæmatoma was followed by the local formation of an abscess which required a further, also successful operation.

Where there are no external indications of the site of the hæmorrhage, it can often be inferred from the symptoms, and recovery may be brought about by clearing out the blood clot. Operative treatment then is particularly indicated when the symptoms of cerebral compression become threatening and the local symptoms show no tendency to diminish. I have, however, seen a few cases of this kind in which the symptoms of compression as well as the local signs spontaneously improved, even in a case in which operation had been decided on on account of the increasing cerebral compression. On the other hand trephining has frequently been without result because the hæmorrhage has had its site, not in the meninges, but deep in the substance of the brain (Walton-Brooks and others). H. W. Page is strongly in favour of operative measures, as are also Enderlen and others.

Hæmatoma of non-traumatic origin has also occasionally been treated surgically (Michaux, Jaboulay, Neisser, etc.: see previous section).

In some cases of meningeal hæmorrhage the recovery has been attributed to a single or frequently repeated lumbar puncture (Rochard, Potherat). Tuffier and Quénu (ref. *C. f. Gr.*, 1906) speak very warmly in favour of this method. Devreigne (*Presse méd.*, 1905) advocates the procedure in infants, born apparently dead from injuries received during birth, while Cushing (*Amer. Journ. of the Med. Sc.*, 1905) from personal experience is in favour of immediate operative treatment.

Acute Inflammation of the Lepto-meninges

Acute (Purulent) Cerebral Meningitis or *Lepto-meningitis*

Summaries of the literature, including at least the more important treatises, will be found in the following: F. Schultze, Nothnagel's "Handbuch," ix.; Körner, "Die otit. Erkr. d. Hirns., d. Hirnhäute," etc., III. Aufl., 1902, and "Nachträge" zur III. Aufl., Wiesbaden, 1908; Leyden-Goldscheider, Nothnagel's "Handbuch," x.; Henoch, "Lehrbuch der Kinderkrankheiten"; Brieger, "Die otog. Erkr. d. Hirnhäute," *Würzb. Abhandl.*, 1903; Kohts, *Deutsche Klinik*, etc., vii.; Hölscher, "Die otog. Erkr. d. Hirnhäute," Halle; Royet, *These de Paris*, 1905; Barnhill, *Journ. of the Amer. Med. Assoc.*, 1905, and Discuss.; Heine, *B. k. W.*, 1900; H. Stroebe, "Handbuch d. path. Anat. d. Nerv.," Bd. i. Further references in the text of this chapter.

Acute inflammation of the fine cerebral membranes has, as a rule, a *diffuse* distribution. Although it may be most marked over the base of the brain, or over the convexity, it is seldom entirely limited to the one or other of these sites. This disease, starting in the pia-arachnoid, usually affects the brain also, especially the cerebral cortex, so that in most cases one may speak of a *meningoencephalitis*.

Etiology.—Purulent meningitis is due to micro-organisms. Streptococci and staphylococci may be found in the exudate, but certain special species to which a specific influence must be attached, *e.g. Fraenkel's pneumococcus* and the *meningococcus intracellularis of Weichselbaum*, may be present. According to the investigations of Leyden, Jaeger, Netter, Heubner, Fürbringer, Longo, Lingelsheim, Councilman, and others, the

latter of these seems to be the special agent at work in epidemic cerebrospinal meningitis (*q.v.*), but in purulent meningitis, Fraenkel's pneumococcus, Friedländer's pneumobacillus, B. typhosus, Pfeiffer's bacillus (Mya, Jundell, Hecht), B. coli, the Bacterium lactis aërogenes, etc., have been found, so that the question of the specific significance of the various infective agents cannot be regarded as finally settled. Experimentally, meningitis has been successfully produced by the introduction of Fraenkel's pneumococcus and the meningococcus intracellularis (Netter, Foa, Fraenkel, H. Bruns, Flexner).

The micro-organisms may reach the meninges from a *neighbouring area of suppuration*, or they may come by way of the blood (or lymph) stream as part of a *general infection*, or be carried to the brain from a *distant seat of infection*. *Injuries* to the skull with open wounds—apart from those directly involving the meninges—may lead to meningitis, as the infective germs produce suppuration in the neighbourhood of the wound, which is transmitted to the meninges. A sinus thrombosis spreading from the veins of the diploë may play an intermediate rôle in this process.

According to the experiments of Ehrenrooth (*Z. f. N.*, xx., and *Acta Scient. Fennicæ*, 1902) injuries to the head, not producing wounds, may lead to meningitis if at the same time pathogenic micro-organisms are circulating in the blood. The observations of Huismans, H. Curschmann (*D. m. W.*, 1904), and others point in the same way.

It would seem to follow from the observations of Fujisawa (*M. m. W.*, 1901), Nonne (*N. C.*, 1907), Bontemps, and others that purulent meningitis may develop a long time after injury to the skull. Bayerthal (*N. C.*, 1905) describes an interesting case of this sort which is difficult to interpret, likewise Graf (*Charité-Annalen*, xxvii.). Nonne and Bontemps describe meningitis due to an encapsulated bullet.

Injuries which open into the vertebral canal may lead to purulent cerebrospinal meningitis, and bedsores, with erosion of the sacrum, may also allow meningitis-producing organisms to enter the vertebral canal. In recent times operative treatment of vertebral and spinal cord diseases and lumbar anæsthesia (Sonnenburg, Oelsner, and others) have repeatedly been the starting-point in the development of a purulent meningitis.

Meningitis not infrequently results from erysipelas of the head and it is occasionally due to *actinomycosis*.

The ear is a particularly common site of origin. Purulent *otitis* and *caries of the petrous temporal bone* form one of the most important and common causes of cerebral meningitis. The purulent material gains access to the interior of the skull through the thin tegmen tympani or through the roof of the mastoid antrum, or it may spread along the nerve sheath of the seventh and eighth cranial nerve to the meninges. Thrombosis in the veins and sinuses is not infrequently the connecting link between disease of the ear and meningitis. Old people, according to Heine, are specially liable to otitic meningitis.

Inflammation of the *nasal cavities* of the *orbit*, the sinus frontalis, or the pharynx much less often extend to the cerebral membranes. But a purulent meningitis may result from operative measures in these areas and may even follow simple probing of the accessory cavities of the nose, and occasionally extraction of polypus or curetting of adenoids (Booth). Naturally also intracranial foci of inflammation (cerebral abscess, extra-dural inflammation) may come to involve the meninges.

Epidemic cerebro-spinal meningitis is to be regarded as a *primary infective disease*.

Suppurative meningitis may further result from *pymia*, *septicæmia*, and *acute infective diseases* (pneumonia, typhoid, ulcerative endocarditis, small-pox, influenza, acute articular rheumatism, gonorrhœa (?), etc.).

In rare cases injuries to a distant part of the body may give rise to a metastatic meningitis without any other evidence of a pyæmic or septic infection being present (E. Levy).

Its connection with intestinal catarrh is still very doubtful.

It is highly improbable that *sun-stroke* can cause a purulent meningitis, nor has it been proved that simple concussion or mental over-exertion can bring about this condition.

In many cases the etiology is obscure. There is a form of suppurative meningitis occurring sporadically which corresponds in its course to epidemic cerebro-spinal meningitis, and probably has the same (infective) origin. It is convenient, however, to refer to epidemic cerebro-spinal meningitis and to tuberculous basal meningitis in a special section, and to deal here with the other forms of acute meningitis.

Pathological Anatomy.—Purulent—non-tubercular—meningitis extends by preference, though by no means exclusively, over the convexity of the brain. There is first of all hyperæmia of the pia mater, which later becomes cloudy in appearance. The exudate is not always purulent from the onset, and especially when the meningitis has followed on a head or spinal operation, the previously serous cerebrospinal fluid becomes at first turbid and then takes on a purulent character. Purulent meningitis due to ear disease undoubtedly often goes through a serous stage. At the commencement the covering of pus is found mainly over the sulci; localised collections of pus develop, and these become confluent, until a thick layer of greenish yellow pus covers the whole membrane, especially over the convexity. Changes are likewise found in the superficial layers of the cerebral cortex: serous transudation, encephalitic processes, small hæmorrhages, and collections of pus are the anomalies occurring here. We cannot here describe the finer changes of the cerebral cortex, which have been studied recently, particularly by Thomas,[1] Faure, and Laignel-Lavastine.[2] Abscesses rarely form in the interior of the brain: areas of softening have been found. A sero-purulent exudation into the ventricles, and even a considerable degree of hydrocephalus is an ordinary feature of meningitis. It is usually due to the closing of the opening between the ventricle and the sub-arachnoid space, by the meningitic process. A purulent meningitis, limited to the cerebral ventricle, may occasionally though very rarely occur in childhood. There are also forms of circumscribed purulent meningitis, limited to one hemisphere, or even to one lobe; they are generally either otogenic or rhinogenic in character.

The meningitis usually extends to the *spinal meninges*, although it is less pronounced here; it may, however, spread quite to the lower end of the cord. When the suppuration begins in the spinal meninges it may extend gradually or very rapidly towards the brain (Lichtheim). Otogenic meningitis may be almost limited to the spinal membranes.

In exceptional cases one finds a hæmorrhagic form of purulent meningitis with small and larger effusions of blood into the membranes. Cases of this kind have been recorded by Clauss-

[1] "Les altérations du Cortex dans les Méningites aigues," Paris, 1902.
[2] *R. n.*, 1902; *Arch. gen. d. Méd.*, 1904.

nitzer (*Inaug. Diss.*, Leipzig, 1900), Eichhorst (*V. A.*, 1898), Risel (*Z. f. Hyg.*, 1903), Renvall (*Arbeit. Inst. Homén, q.v.* for literature).

F. Schultze[1] has shown that one may have a meningitis *sine* meningitide a brain disease developing from infection with the clinical features of meningitis, with no pathological changes, or only trifling ones in the meninges, but with round-cell accumulations in brain and cord. Finkelstein has noted similar appearances, and following Seitz he attributes the clinical features to the presence of organisms or of their toxins in the cerebro-spinal fluid. A case of Birnbaum's[2] is particularly interesting in this connection, as the meningococcus intracellularis was found in the cerebro-spinal fluid while the post-mortem findings were completely negative. The meningitis may, further, be so fulminating that there is not time for pus to form (Klebs, Leichtenstern).

Wicardt refers to fulminating meningitis in cases where the injection has come from the ear (*Progrès méd.*, 1907).

Symptomatology.—Although the symptoms vary according to the site, extent, and nature of the meningitis, we can draw up a clinical picture which will include all the typical cases.

The commencement of the disease is often obscured, inasmuch as it follows on some other affection with cerebral features, *e.g.* a head injury, an infective disease, etc. Where it begins clearly, it is characterised as a rule by the onset of *headache*, severe and constant but with exacerbations. This headache is generalised, though sometimes specially felt in the frontal and occipital regions. In the course of a few days there follows *dulling of consciousness*; the patient becomes unintelligible and delirious, first during sleep, then permanently, or there are alternating periods of *somnolence* and wild *delirium*. It is characteristic that the headache persists during the delirium, that the patient sometimes cries out loudly from pain during his confusion and stupor. Even when stuporose he grasps his head and contorts his face painfully, especially when one tries to move the head passively. So long as he is sensible, he complains also of *giddiness* and sensitiveness to light and sounds. This *hyperæsthesia* is sometimes still manifested in the unconscious stage by the marked shrinking from noises, touch, etc.

Vomiting is frequently, although not always present; it conforms to the characteristic cerebral type.

Fever is present as a rule from the commencement. The temperature may rise to 40° C. during the first days and remain at this height till death, but generally there are irregular variations within wide limits. Before death a further rise, even to 42° C., is to be noted. Not infrequently, however, the temperature may fall far below the normal. Rigors may be present either at the beginning or during the course of the disease. The *pulse* is as a rule rapid, but sometimes pulse and temperature are disproportionate, in that the former is not accelerated correspondingly with the degree of the fever. At the commencement especially it may be *slowed* to fifty or less per minute. One of the most important signs of meningitis is *stiffness of the back of the neck*, which develops sometimes at the very start, sometimes during the course of the disease. It may be attributed to meningitis of the posterior fossa, and is hence a constant symptom of

[1] "Verhandl. d. vi. Kongr. f. innere Med.," 1887. [2] *M. m. W.*, 1903.

basal meningitis. It is especially well brought out on attempting to make the patient sit up. A certain degree of rigidity often affects the muscles in the upper part of the body also. If the patient is placed in a sitting position, so that the legs hang over the edge of the bed, the tightness of the hamstring muscles becomes markedly noticeable and the leg cannot be fully extended (Kernig); and, as in Lasègue's sign, if the knee is extended when in the dorsal position, complete flexion at the hip is impossible and the attempt causes pain. Netter [1] found this symptom, to which he attaches great importance, in forty-one cases out of forty-six, and it was evident when other features were very poorly developed; Roglet and also Chauffard have had similar results. Jansen and Körner [2] allude to its diagnostic importance in otitic meningitis. The sign, which may also be found in typhoid, uræmia, etc., and may be absent in meningitis (Oppenheim, Clark, Abadie,[3] Wennagel,[4] Willson,[5] and others), cannot, however, be regarded as in any way pathognomonic, as Kernig himself now states; nevertheless in acute cases the symptom points with great likelihood to meningitis. The abdominal muscles are often board-like and the abdomen is retracted and scaphoid.

Hyperæsthesia of the skin and muscles is usually present. Quite light touches bring out vigorous reflexes, although the abdominal reflexes may disappear in the later stages. Tapping the lumbar muscles causes spasmodic indrawing of the vertebral column (back-sign, as I have called it). The *vasomotor* excitability is usually also increased, stimulation of the skin brings about intense and lasting redness, although this symptom is in no way characteristic. Herpes and other skin affections only occur in exceptional cases of non-epidemic meningitis. Herpes labialis has been noted by Körner and Schulze in otitic meningitis.

In addition signs appear which indicate involvement of the *brain* and *cranial nerves*. The latter are the more important and constant, and are specially evident when the meningitis extends over the base of the brain. The *nerves to the eye-muscles*, the *facial* and the *optic nerves* are most of all affected. We therefore often quite early find some difference in the size of the pupils; at first they are usually contracted, later dilated. *Loss of the pupillary reflexes* also occurs. Mydriasis may be present, one day in the left eye, and in the right the next. *Ptosis* and paralysis of one or more of the external eye muscles, with corresponding squints, are frequently observed; less frequently nystagmus. A very noteworthy but by no means constant sign is *optic neuritis*, which is often only partially developed and seldom reaches the stage of complete choked disc. The visual acuity is as a rule but slightly diminished thereby, although naturally it is difficult or, it may be, impossible to prove this.

According to the experience of Pitt (*Brit. Med. Journ.*, 1890) and Körner, optic neuritis is a rare phenomenon in uncomplicated acute otogenic leptomeningitis.

With regard to further ocular complications in purulent meningitis the reader is referred to the following chapter.

Paresis of one facial nerve occurs not infrequently. It is generally preceded by *slight twitchings* of the face muscles, or these may appear after the paresis has set in. It is quite commonly associated with reaction of degeneration, although I have only once succeeded in obtaining it

[1] *R. n.*, 1900. [2] "Die otit. Erkr. Nachtrag.," 1908. [3] *R. n.*, 1903.
[4] *A. f. kl. M.*, Bd. lxxxvii. [5] *Amer. Journ. of Med. Sci.*, vol. cxxx.

myself, as the disease mostly ends fatally before the change in the reaction has had time to develop fully. *Trismus* is sometimes present. Irritant and paralytic symptoms in the domain of other cranial nerves have very rarely been noted.

Involvement of the cerebral cortex is made manifest by irritant and paralytic phenomena, but generally it is only the affection of the motor zone which finds clinical expression. We have *slight twitchings* in the extremities and *unilateral* or *general convulsions*; contracture of the limbs of one side of the body is sometimes present also. Paralysis, *monoplegia* or *hemiplegia*, becomes evident later on, or it may be from the outset—or more rarely there is a central facial paralysis (limited to the lower face). *Aphasia* is common in tubercular basilar meningitis, but is only exceptionally observed in the other forms, as *e.g.* in a case of Kuhn's of *otitic* meningitis. The presence of hemianopsia has scarcely ever been affirmed with certainty in the acute forms of meningitis. A unilateral hyperæsthesia is sometimes present, hemianæsthesia scarcely ever.

The tendon reflexes are usually exaggerated at first, but later on they tend to disappear. One is not entitled, without further evidence, to attribute this to the spinal meningitis, as high fever and rise of pressure in the cerebro-spinal fluid may quite well bring about disappearance of the knee-jerks; this corresponds to the fact that in some cases they are only temporarily lost.

There is usually *constipation*. *Retention of urine* is common in the stuporose stage; more rarely there is *incontinence*. The scanty urine may contain some albumen. Glycosuria has been observed, but very rarely.

We are indebted to Grunert (*A. f. Ohr.*, Bd. xlix. and lvii.) and Ulrich (*Deutsche Klin.*, xi.) for observations, dealing chiefly with suppuration in the fourth ventricle.

The patient's strength falls rapidly. *Bedsores* may form, especially in the last stages. *Deep coma* with increased reflex excitability is then present. The pulse is small and very rapid. The breathing, which may be irregular at the very onset of the disease, is accelerated and superficial, and may display or develop the character of Stokes breathing. Death ensues in deep coma, sometimes with convulsions.

The *course* of the disease is always *acute* and may be so rapid that death occurs in about forty-eight hours. More usually, however, it extends over one to two weeks, rarely longer. In a case observed by Bergmann, nevertheless, the patient was still alive three weeks after the time that staphylococci were found in the fluid obtained by lumbar puncture. Since signs of irritation are most prominent at first and those of paralysis later, an attempt has been made to differentiate two corresponding stages, but the distinction cannot usually be maintained, as paralytic phenomena may be present from the very onset.

But the clinical features of purulent meningitis are *exceedingly variable* and may differ in almost every particular from the type described, and particularly where it follows on some other disease, as it may then run a completely latent course (Huguenin, Fraenkel, Jansen, Sicard, and others). There are cases in which fever is absent or is merely trifling in amount; others in which there is no stiffness at the back of the neck. There are even cases, although these are rare, where the mental faculties remain clear to the end. The stage of invasion may be *long drawn out*,

even for several weeks. This applies especially to circumscribed purulent meningitis, in which, by gradual or recurrent infections of the originally exempted portions of the meninges, an intermittent character may be given to the course of the disease (Brieger).

Attempts to relate the varying symptomatology of meningitis to differences in the exciting agents, have so far led to no very satisfactory result.

Amongst the most frequent *complications* of this disease are *sinus thrombosis* and *brain abscess*.

The *prognosis* of purulent meningitis is very gloomy. But, just as in epidemic cerebro-spinal meningitis the possibility of cure of a similar process has been demonstrated, so in recent times the cases in which a meningitis, claimed to be purulent, of otogenic or other origin, has ended in recovery have greatly multiplied. Many of these cases were undoubtedly often not the purulent, but the serous form of meningitis—the preliminary stage of the former (Körner), but there remain over and above, cases in which the purulent character has been established by splitting the dura mater or by lumbar puncture (see below) (Netter, Gradenigo,[1] Bertelsmann,[2] Langer, Jansen, Nobécourt, Provost,[3] and others). These results, however, require revision, inasmuch as further experience (see below) has shown that lumbar puncture affords no absolutely certain conclusion as to the presence of a general purulent meningitis. In otogenic meningitis recovery sometimes follows on the pus bursting out through the ear, but confusion with cerebral abscess is possible here. In recent times Fischer [4] has reported a case of this sort. This termination is most likely in the localised form. We are indebted to Gaussel for an interesting communication of this kind. Recoveries have also followed in traumatic and operative meningitis spreading upwards from the spinal cord (Barth, Schultze-Schede). Gowers refers to two cases of meningitis following on puerperal sepsis, with a favourable termination. If deep coma has set in, maintenance of life can scarcely be hoped for. One must be cautious in giving a prognosis, however, so long as the diagnosis is in any doubt.

The fact that the prognosis is especially bad in pneumococcal meningitis, has been recently insisted on (Kaupe and others), and Körner is of the same opinion with regard to streptococcal meningitis.

Hydrocephalus may persist after complete recovery.

Differential Diagnosis.—Although the disease is readily recognised in typical cases, under other conditions the diagnosis may present the greatest difficulty. It happens, not infrequently, that an acute infective disease, especially *pneumonia* and *typhoid*, accompanied by cerebral symptoms, is mistaken for meningitis. The symptoms leading to this error are headache, delirium, stupor, fever, and perhaps vomiting. In doubtful cases one ought not to make a diagnosis of meningitis from these symptoms alone. Special caution is required in childhood, as many children become delirious and have convulsions with any rise of temperature, so that even a simple sore throat has at first been taken to be

[1] *A. f. Ohr.*, Bd. xlvii. [2] *D. m. W.*, 1901.

[3] "Formes curables de Méningite," etc., *Thèse de Nancy*, 1901-2. See also Lannois-Perretière, *Lyon méd.*, 1906.

[4] *Prag. med. Woch.*, 1903.

meningitis. A careful examination of the lungs will prevent confusion with pneumonia, while the presence of an enlarged spleen, the roseolar rash, the characteristic stools (in meningitis there is usually constipation), the peculiar behaviour of the temperature, the presence of typhoid bacilli and the Gruber-Widal reaction, etc., permit us to recognise typhoid. The onset of typhoid, also, is gradual, while that of meningitis is acute, and—if we except the tubercular form—without premonitory symptoms. In any case one should only diagnose meningitis in cases of this sort when the pathognomonic symptoms—stiffness at the back of the neck (which, it is true, also occurs as a premonitory symptom in typhoid), paralysis of some of the cranial nerves, optic neuritis, cortical phenomena, etc.—are present. Headache persisting throughout the delirium and the stupor is always suggestive of meningitis. Slowing of the pulse despite the raised temperature must also raise the suspicion that one is dealing with a cerebral disease. Kernig's sign cannot be regarded as a certain differential feature, a point upon which Netter and Carrière have specially laid stress. In doubtful cases lumbar puncture (see below) may help to confirm the diagnosis. Whether and how far a knowledge of the *opsonins* will be of aid in the differential diagnosis the future will show. *Septicæmia* and *pyæmia* are likewise accompanied by cerebral symptoms, which may simulate the picture of a meningitis ; but here also the progress of the case fails to show any sign of cranial nerve involvement or of neck stiffness. As we have mainly to distinguish between septicæmia and meningitis, joint-swellings, phlegmonous processes, cutaneous and retinal hæmorrhages, repeated rigors, etc., are in favour of the former. Careful examination will also reveal some source of the septicæmic process. Examination of the blood rarely yields positive information by the presence of a pus-forming agent.

Acute purulent otitis is sometimes, especially in children, accompanied by cerebral symptoms which in many respects correspond with those of meningitis. These are headache, giddiness, and stupor. In children there may even be delirium and general convulsions, and though it is difficult to understand, optic neuritis has occasionally been recorded. An abducens paralysis may also result from direct spread of the inflammatory œdema (Körner, Peyser, Terson). All these phenomena may vanish with the evacuation of the pus or the subsidence of the otitis. It is not yet quite clear what is the relationship between diseases of the ear and cerebral affections which are very like meningitis, but which result in complete or incomplete recovery (with permanent visual disturbance or even optic atrophy). So-called meningitis serosa (*q.v.*)—the precursor of meningitis purulenta, as Körner called it—is apparently for the most part responsible for the symptoms. The importance of lumbar puncture in the differential diagnosis of these affections will be referred to afterwards. Extra-dural abscess in the posterior cerebral fossa may also give rise to a clinical picture which in many respects corresponds to that of meningitis (comp. the chapter on brain abscess).

Uræmia can practically always be recognised from examination of the urine, the presence of œdema, etc. It is true that albuminuria may also occur with meningitis, but tube-casts, etc., are absent. In doubtful cases the further progress soon elucidates the condition.

Tschteglow has seen clinical features resembling those of a fulminating meningitis in anthrax from bacillary emboli in the cerebral vessels.

Syphilitic meningitis may also run an acute course. There may be very occasionally a varying degree of pyrexia. In one such case, in which ptosis, difference in size of pupils, stiffness of neck and stupor were the chief symptoms, I observed rapid recovery from treatment with potassium iodide. We are indebted for interesting but not quite unequivocal observations of this kind to Widal and Le Sourd,[1] Brissaud-Brécy,[2] and Sabrazès.[3]

It is still necessary to refer also to a number of conditions which have been described by various authors under the heading *pseudomeningitis*, *méningisme*, etc. (Kohts, Dupré, Seitz, Lépine, Donath, Lamuroux,[4] Krannhals,[5] Rocca,[6] Galliard, Dauchez,[7] Finkelstein, Leichtenstern, Maixner-Simerka, Jackson,[8] Taillens,[9] Stursberg,[10] and others). They deal with symptoms and groups of symptoms which are closely allied to those of meningitis, but the final recovery, the results of post-mortem examination or examination of the fluid obtained by lumbar puncture, show that there has been no underlying purulent inflammation of the meninges but either no change whatever or merely œdema, hyperæmia, or possibly serous effusion. In one group of these cases a toxic brain disease is probably the underlying condition, in another the course is so rapid that there is no time for pus to form, and lastly, the so-called serous meningitis (see chapter on acquired hydrocephalus) may give rise to a group of symptoms allied to those of purulent meningitis. These observations warn one to be very cautious in making a diagnosis of purulent meningitis, and such caution is particularly necessary where the cerebral features develop during the course of an acute infective disease (especially *pneumonia* and *influenza*) or after one of these, and where they deviate markedly from the typical meningitis described above. Even under these conditions a real meningitis may underlie the clinical picture of a pseudo-meningitis as has been shown by lumbar puncture, *e.g.* in a case of Langer's, and as follows from the observations of Sacquépée-Peltier and others.

Voisin (*Rev. mens. des mal de l'Enf.*, 1904), states that his systematic histological investigation shows that in pneumonic processes in children inflammatory changes are as a rule present in the meninges.

Non-purulent meningitides likewise occurs in otitis (Levi, Lecène-Bourgeois,[11] Joel,[12] Kretschmann,[13] Oppenheim, Lucae, Jansen, Waldvogel,[14] R. Müller, Hegener,[15] Hammerschlag,[16] O. Brieger, and others). They may also originate from the nose (Herzfeld[17]).

Further, I have seen a serous form of meningitis resulting from the pressure of a bullet on the wall of the lateral ventricle.

This *meningitis serosa* is dealt with in the chapter on hydrocephalus.

We will not here discuss the meningeal process developing in connection with non-purulent encephalitis (*q.v.*), and will merely mention the so-called adhesive arachnitis which has been described by Krause-Placzek (*B. k. W.*, 1907).

In alcoholics meningitis may be confused with *delirium tremens* and

[1] *Bull. et Mém. de la Soc. méd. de Paris*, 1902.
[2] *R. n.*, 1903.
[3] *Gaz. hebd. de Bordeaux*, 1903
[4] *Thèse de Paris*, 1902.
[5] *A. f. kl. M.*, Bd. liv.
[6] *Thèse de Paris*, 1898.
[7] *Rev. mens. des mal. de l'enf.*, 1899.
[8] *Journ. of Amer. Med Assoc.*, 1907.
[9] *Arch. de Méd. des enf.*, ix.
[10] *Z. f. N.*, xix.
[11] *Presse méd.*, 1902.
[12] *D. m. W.*, 1895.
[13] *M. m. W.*, 1896.
[14] *D. m. W.*, 1898.
[15] *M. m. W.*, 1901.
[16] *W. m. W.*, 1900.
[17] *B. k. W.*, 1905.

meningitis may produce a real alcoholic delirium so that its symptoms are temporarily concealed. In delirium tremens the stiffness of the neck, intense headache, and, in particular, the focal symptoms are lacking. When the patient—as often happened, *e.g.* at the Charité—is brought to hospital in the last stages, or when the meningitis takes a maniacal course, it may quite possibly remain unrecognised.

In children weakened by gastro-intestinal catarrh, some of the symptoms of meningitis—somnolence, convulsions, trismus, etc.—may occur. In these conditions, described as hydrocephaloid, there is no fever, however, the focal symptoms and the optic nerve affections are lacking, and the etiology, sinking in of the fontanelles and accompanying appearances, will settle the diagnosis. *Gastro-intestinal autointoxication* in childhood may also give rise to conditions which clinically closely resemble meningitis (Jaksch, Carrière,[1] Stuertz,[2] and others). The gastric symptoms, the presence of indican in the urine, and the rapid results of clearing out the bowel (by calomel, enemata, etc.) allow one to distinguish these conditions from meningitis. Finally, lumbar puncture may aid in their differentiation.

It has also been asserted (Fesca, Marco [3]) that ascarides may give rise to conditions of intoxication which are similar to those of meningitis (helminthiasis meningitiformis).

Finally, it happens that *hysteria* gives occasion for diagnostic errors, as it may cause delirium, convulsions, stupor, and also stiffness of the neck. Some time ago, I was called to a patient who, on account of these symptoms, had already had her head closely shaven and rubbed with tartarated antimonial ointment, while after a few hours she was able to leave her bed. French authors especially (St Ange-Arnozan,[4] Huchard,[5] Pitres,[6] Chantemesse,[7] Macè,[8] and others) have described cases of *pseudo-meningitis hysterica* in which the clinical picture closely resembles that of purulent or tubercular meningitis. Stiffness of neck, vomiting, rigors, delirium, coma, paralysis, even squint, loss of pupil reflexes, and slowing of the pulse are said to have been observed (?). Here the aetiology is the chief point to be kept in mind. The known causes of meningitis are absent, whilst the symptoms almost always directly follow upon some preceding psychical irritation. The symptoms are hysterically coloured and hysterical stigmata are usually present. With these hysterical attacks there is either no rise of temperature or one which does not correspond with the fever of meningitis. The pupils are almost always equal and react normally; optic atrophy, facial paresis, etc., are never present. The eyes are usually kept spasmodically closed. The so-called *acute fatal hysteria* is such a rare affection that the differential diagnosis scarcely requires consideration.

H. Starck (*Z. f. N.*, xxi.) has described a very interesting case of psychogenic pseudomeningitis, although it is not of great value for differential diagnosis, as the patient, a former hospital attendant, was not only hysterical but a cunning impostor. Among the features of his case were fever, slow pulse, Kernig's sign, and even rise of pressure in the cerebro-spinal fluid; the case indeed had given occasion for the performance of laminectomy, etc.

In recent years we have acquired a new diagnostic method in *lumbar*

[1] *Le Nord méd.*, 1902.
[2] *B. k. W.*, 1903.
[3] *Allg. W. med. Z.*, 1902.
[4] *Gaz. méd. d. Bordeaux*, 1893.
[5] *Revue gén. de Clin.*, etc., 1890.
[6] *Leçons cliniques sur l'hystérie*, 1891.
[7] *Thèse de Paris*, 1884.
[8] *Thèse de Paris*, 1888.

puncture[1] (Spinalpunktion, Lendenstich). This method, founded by Quincke (1891), consists essentially in puncture of the vertebral canal and the withdrawal of a small quantity of the cerebro-spinal fluid contained in the sub-arachnoid space of the spinal cord which is then submitted to physical, chemical, microscopic, and bacteriological examination.

Quincke recommends the following procedure: The patient lies in the left lateral position, well bent forwards at the lumbar portion of the vertebral column. The space between the third and fourth or between the fourth and fifth lumbar vertebræ is selected, then, in the middle line in children, 1 cm. from it in adults, at the level of the lower border of the spinous process, the needle is pushed in slightly upwards and towards the middle line until the cessation of resistance shows that the point has penetrated into the sub-arachnoid space. To measure the pressure the canula should be connected by india-rubber tubing with a narrow bent glass tube or with a mercury manometer.

There are many modifications of the method and the apparatus. First of all as regards the site of puncture, others recommend the space between the arches of the 2nd and 3rd lumbar vertebræ, and Chipault, with whom Fürbringer agrees, selects the sacro-lumbar hiatus, the space between the last lumbar vertebra and the upper border of the sacrum. Concetti recommends the level of the line joining the two posterior iliac spines. Schönborn chooses the space at or just below the spot where the horizontal line joining the iliac crests cuts the vertebral column. Whether one should introduce the needle directly, in the middle line, or somewhat laterally to it, at the level of the lower border of the dorsal process or somewhat higher, depends mainly on the individual peculiarities of the spinal processes; on the whole it is advisable to follow Quincke's instructions, and only to modify the procedure by moving the needle or by choosing another spot should one meet with bony resistance. The needle must be pushed more or less deeply in according to the thickness of the panniculus adiposus and other parts which it has to go through; this varies from about 2-7 cm. One must not be timid in this connection, however, as I know from personal experience. The question of lumbar-puncture has been studied by the following, in addition to the writers already mentioned: Lenhartz, Rieken, Wilms, Krönig, Goldscheider, Strauss, Pfaundler, Ranke, Neurath, Leutert, Cassel, Nawratzky-Arndt, Mya, Widal, Sicard, Levi-Sirugue, Roqueta, Archard, Camille Wolf, Descos, Pellagot, Léri, Babinski-Nageotte, Devaux, Rhodes, Schönborn, Campbell-Warrington, Nissl, Chavasse-Mahu, and others.

Quincke uses a canula of ½-1mm. diam., provided with a fine wire to clear it. Braun prefers a hollow needle. Schönborn recommends a hollow platinum-iridium needle 10-12 cm. in length (obtainable from Galante, Paris). Quincke's apparatus has been modified by Krönig, especially with regard to pressure measurement, the former only permitting this to be done after several cc. of fluid have been withdrawn, while Kronig's apparatus with its capillary upright tube overcomes this difficulty and allows the pressure to be measured as soon as a very small quantity has escaped. I agree so little with Krönig regarding the extent of the indications for this operation that I am all the more bound to acknowledge the merits of his apparatus. An Hg-manometer is not necessary; a V-shaped glass tube, of which the ascending limb has sufficient length (about

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[1] For the literature on this question see Quincke, *B. k. W.*, 1891 and 1895; *ibid.*, "Die Technik der Lumbalpunktion," 1902; *ibid.*, "Die diagn. und therap. Bedeut. d. L.," *D. m. W.*, 1905; Lichtheim, *B. k. W.*, 1895; Chipault, *Annales de la Orthop.*, 1896; Fürbringer, *B. k. W.*, 1895; Finkelstein, *Charité-Annalen*, 1898; Stadelmann, *B. k. W.*, 1895, and *D. m. W.*, 1897; *Mitt. aus d. Grenzgeb.*, 1897; Neurath, "Sammelref," *C. f. Gr.*, 1898; Pfaundler, Wien, 1899; Sicard, "L'Encyclop. scientif. des Aide-Mémoire," ref. in *R. n.*, 1902; Krönig, "Verhandl. d. xvii. Kongr. f. inn. Med.," Dermat C., 1905, and *D. m. W.*, 1897. References to most of the French works in *R. n.*, 1902-1907; Léri, *Arch. de méd. des enf.*, 1902; Lewkowicz, *Jahrb. f. Kind.*, Bd. lv.; Devaux, *C. f. N.*, 1903; Schönborn, *N. C.*, 1903; Campbell, *R. of N.*, 1904; Nissl, *C. f. N.*, 1904; Dana, *Med. Record*, 1904; Gerhardt, *Mitt. aus d. Grenzgeb.*, xiii; Merzbacher, *N. C.*, 1904; Wertheimber, *M. m. W.*, 1904; Chauffard-Boidin, *Gaz. des hôp.*, 1904; Niedner-Mamlock, *Z. f. k. M.*, liv, Mott; *Brit. Med. Journ.*, 1904; Skoczynski, Diskuss, *N. C.*, 1905; Fuchs-Rosenthal, *W. m. P.*, 1904; Rehm, *C. f. N.*, 1905; Balogh, *W. m. W.*, 1906; Raubitschek, "Die Zytol. der Ex- und Transsud. Sammerlref. mit Lit.," *C. f. Gr.*, 1906; Schönborn, "Volkmanns Samml.," *N. F.*, 1905, and *Med. Kl.*, 1906; Stewart, *Ed. Med. Journ.*, 1906; Apelt, *M. f. P.*, xx. Suppl.; Zupnik, *Prag. m. Woch.*, 1906; Benischek, *B. k. W.*, 1906 (ref.); Buzzard-Batten-Forbes, *Brit. Med. Journ.*, 1907; Henkel, *A. f. P.*, Bd. xlii.; Cimbal, *Therap. d. Geg.*, 1906; Nonne-Apelt (*A. f. P.*, Bd. xliii.).

½-1 metre) is all that is required. Neither is it necessary or even advantageous to fill the tube at the outset with sterile saline solution (Stadelmann).

It is sufficient for diagnostic purposes to draw off a small quantity of the cerebro-spinal fluid. Above all it is necessary when doing this to control the pressure, and if it sinks rapidly to stop the outflow. The pressure should not be allowed to fall below 50 mm., in cases of very high original pressure not below 300 mm. The fluid should only be allowed to drop out slowly and regularly. Pulsatile oscillation indicates that there is free communication between the sub-arachnoid spaces of the brain and cord (Krönig). Aspiration must be unconditionally avoided.

If the outflow is stopping, H. Curschmann ("Therapie d. Gegenw.," 1907) recommends the application of a cold spray to the thigh as an artificial means of raising the pressure in the fluid.

It is hardly necessary to say that the little operation is to be carried out antiseptically (heating the needle thoroughly, etc.). Narcosis is practically always unnecessary; local anæsthesia by cold spray or infiltration is sufficient. Only in mental cases has Nissl occasionally had to employ a general anæsthetic. Quincke's advice, to keep the patient quietly in bed for 24 hours after the puncture, should be acted upon in every case.

According to Quincke the normal pressure amounts to 40-60 mm. of water, and he regards it as pathological only when it exceeds 150 mm. According to Krönig, it is on an average 125 mm. when the individual is lying on his side, but it varies from 100-150 mm. in different persons. In a sitting posture it amounts on an average to 410 mm. Under pathological conditions the pressure may rise to 700 mm. or even more. In general one may also regard the pressure as increased when the fluid comes out in a steady stream. According to Krönig the fluid flows in drops when the patient is lying down; when he is sitting, on the other hand, it gushes and spurts. The amount which runs off normally varies from a few to 50 c.cm., seldom more, but we have no extensive investigations on this point.

Puncture may yield no information when communication between the vertebral canal and the cavity of the skull is obstructed by closure of the foramen of Magendie, *e.g.* from adhesion and growth of the meninges; in distension of the ventricles also the outflow may be hindered in a purely mechanical way by pressure of the brain against the bones or by pinching of the aqueduct of Sylvius (Quincke), or by the cerebellum and medulla oblongata being jammed in the foramen magnum.

The normal cerebro-spinal fluid is as clear as water, contains little albumin (0·2-0·5 per thousand) and very few cells (none at all according to Widal, and according to Sicard and Schönborn at most 3-4 cells in a field with a magnification of 400).

Guillain and Parant (*R. n.*, 1903) state that the normal fluid contains only globulin, but that under pathological conditions albumin also appears. Nissl was unable to come to any very certain conclusion on this point (see chapter on general paralysis). See also the reports of Skoczinsky, Rehm, Cimbal, and especially of Nonne-Apelt, who give a full description of their method (separation of the albumin by ammonium sulphate).

Pathological conditions are characterised by the following changes: increase in the quantity of outflow, rise in pressure, turbidity of the fluid, admixture of blood, fibrin-clot, and pus, increase in the albumin content (up to 7 parts per 1000 and more), coagulability, and presence of cellular

elements of various kinds as well as of micro-organisms in the fluid. According to recent experiences of Wassermann, Plaut, Citron, and others, the so-called antibodies produced by the action of the organisms and their toxins may also be present.

These factors are of unequal value. First of all it must be insisted upon that increase in the amount of cerebro-spinal fluid and of its pressure occurs under very varied conditions, namely, in cerebral tumour, sinus thrombosis (?), hydrocephalus or meningitis serosa, the various forms of meningitis, and other conditions (uræmia, chlorosis, acute infective diseases). But in none of these affections is the condition constant or always demonstrable. Thus the rise in pressure and increase in the amount of fluid which runs off is often lacking in the meningititides. Investigation in this direction is particularly often without result when communication between the ventricles and the sub-arachnoid space of the brain and cord is interrupted by adhesions, by closure of the foramen of Magendie, etc. Among recent observers, Nissl and Schönborn in particular have declared themselves doubtful with regard to the value of a rise in pressure. The albumin content is increased in inflammatory conditions and in obstruction hydrocephalus (tumour); but on the whole the diagnostic value of this symptom is so far insignificant. Greater importance seems to be attached to it, however, in the diagnosis of the metasyphilitic affections, especially tabes and general paralysis (Monod,[1] Widal, Sicard, Ravant,[2] Nonne, Apelt [3]). Lichtheim and Schiff lay special stress on the coagulability of the fluid on standing as regards the diagnosis of meningitis.

Admixture of blood may of course result from perforation of a vessel on puncture, but this condition is particularly brought about by hæmorrhages into the subarachnoid space of brain and cord and by rupture of a cerebral hæmorrhage into the ventricle. Confusion between blood coming from the subarachnoid space and from a perforated vein is prevented by the fact that the latter coagulates quickly, while the former, when mingled with cerebro-spinal fluid, loses its power of coagulating (Henneberg, Finkelstein). According to Mathieu the blood must have become mixed with the fluid before puncture in cases in which the liquid remains yellow coloured after centrifuging (see in this connection the chapter on pachymeningitis hæmorrhagica). A blackish colouration of the fluid has also the same significance.

The fluid is *turbid*, more especially in *purulent* meningitis; it may also, however, be turbid (although rarely purulent or hæmorrhagic) in the tubercular form. The *purulent* nature is above all characteristic of the various forms of M. purulenta. Even if pus be absent, the large number of leucocytes in the fluid (see below) is a sufficient indication of the purulent character of the inflammation. According to Krönig the cellular elements are distinctly increased in serous meningitis as well as in simple obstruction hydrocephalus and effusions of vaso-motor origin, as compared with normal fluid.

The same author asserts also that he has found in the fluid softened brain matter in the shape of fatty granular cells, myelin, disintegrated nerve fibres, etc., and from these has diagnosed areas of softening in the brain. It is not quite clear to me how these elements can be present in the cerebro-spinal fluid drawn off by careful puncture nor how the rupture through the pia assumed by Krönig comes to pass. In cases of older hæmorrhages he found hæmatoidin crystals. Sabrazès-Muratet (*R. n.*, 1904) give similar accounts. The demonstration by Mott and Halliburton as well

[1] *Thèse de Paris*, 1902. [2] *R. n.*, 1903. [3] *A. f. P.*, Bd. xliii.

as by Wilson (*R. n.*, 1904), that cholin is introduced into the cerebro-spinal fluid by the breaking down of the medullary sheaths, has not yet assumed pathological importance (see, however, the chapter on epilepsy and the corresponding reports of Donath in *Z. f. N.*, xxvii., and Rosenfeld in *C. f. N.*, 1904).

In cases of malignant tumours it may be sometimes possible to find characteristic cells. The suspicion of this to which I referred in the third (German) edition has since been confirmed (Philippe-Cestan-Oberthür, Rindfleisch,[1] Dufour[2]). Cysticercus (Hartmann) vesicles and echinococcal derivatives (?) (Jacob[3]) have each been found in one case.

Bacteriological examination is very important; it must be carried out microscopically in hanging drop preparations and in stained films as well as by culture (perhaps also by innoculation experiments). In purulent meningitis *streptococci*, *staphylococci*, and particularly *pneumococci* or the *meningococcus intracellularis* of Jaeger and Weichselbaum may be present (Lichtheim, Heubner, Fürbringer, Lewkowicz, and others). In the epidemic form the latter is usually present, but Fraenkel's coccus is not infrequently found. Langer demonstrated the presence of Pfeiffer's bacillus in a case of influenzal meningitis, similarly Mya.[4] On the other hand both in influenzal meningitis and other forms of purulent meningitis quite different micro-organisms may be found, *e.g.* B. pneumoniæ, B. coli (the latter by Sacquépée-Peltier, Nobécourt and Pasquier,[5] and others). B. typhosus has occasionally been recorded, as by Stäubli. In tubercular meningitis examination usually reveals the presence of *tubercle bacilli* in the fluid; they are found especially in the clot (it may be necessary to centrifuge), in the majority of cases, according to some authors (Fürbringer, 70 per cent.), according to others, only in a very small percentage (see further reports in the corresponding chapter). The diagnosis may be confirmed by innoculating animals with the fluid (Widal-Le Sourd, Bernheim-Moser).

These results are of special diagnostic value in that micro-organisms are absent in diseases which are allied as regards their symptoms (meningitis serosa, tumour, hydrocephalus, brain-abscess, etc.), although their importance is essentially lessened by their being *inconstant*. Thus in purulent meningitis the transudate may be clear, and free from leucocytes and characteristic organisms (Lichtheim, Stadelmann, Oppenheim, Finkelstein, Achard and Laubry, Schönborn, Witte-Sturm[6]). This also holds good for those cases in which the communication between the brain and the sub-arachnoid space of the spinal cord is cut off. But the exudate may be firm, plastic, and gelatinous, so that the results of lumbar puncture are *nil*; or in consequence of union of the arachnoid with the pia the canula may pass into the sub-dural space (Stadelmann). Finkelstein maintains strongly, however, that the presence of micro-organisms even in clear exudates can frequently be demonstrated by making cultures. If a large quantity of clear fluid flows out it is at all events highly improbable that the case is one of generalised purulent meningitis. On the other hand lumbar puncture has yielded a turbid fluid in occasional cases of brain abscess with circumscribed purulent meningitis (Stadelmann, Wolf,[7] Grunert, Voss,[8] Chavasse-Mahu).

[1] *Z. f. N.*, xxvi. [2] *R. n.*, 1904. [3] *Fortschr. d. Med.*, 1903.
[4] *Gazett. degl. Osped.*, 1903. See also Dubois, *Thèse de Paris*, 1902.
[5] *Rev. mens. des mal. de l'enf.*, xx.
[6] *Z. f. Ohr.*, Bd. xxxix. [7] *Dissert.*, Strassburg. 1897, [8] *Charité-Annalen*, xxix.

From the observations of Grossmann (*D. m. W.*, 1905) it would seem that a small quantity of pus in the fluid is not proof of a general meningitis. Gerber also mentions (*D. m. W.*, 1906) a case of otitic brain abscess cured by operation in spite of the fact that the cerebro-spinal fluid was turbid and contained leucocytes, although not bacteria. See also B. Heine (*B. k. W.*, 1906). From these facts and from his own experience Körner concludes: 1. That in cerebral abscess, sinus phlebitis, or labyrinth suppuration, leucocytes and even bacteria may reach the cerebro-spinal fluid and may remain in it for a considerable time without setting up inflammation in the meshes of the pia (preliminary stage of purulent meningitis); 2. That such a condition may give rise to symptoms which cannot with certainty be distinguished from those of a fully developed purulent meningitis.

The condition of the fluid as to pressure, quantity of outflow, and nature, in the various stages of meningitis, has been studied by means of repeated puncture by Stadelmann, Pfaundler, Councilman, Labré and Castaigne, Griffon and Gaudy, and others. The importance of lumbar puncture in the diagnosis of meningitis, especially the otogenic form, is apparent from the communications of Lecène-Bourgeois, Chavasse-Mahu, Grunert, and others, but we have already referred to the possibility of error. Estimation of the freezing-point (cryoscopy) has so far been of no essential importance in the diagnosis, in spite of the work of Widal, Sicard, Ravaut, Achard, Loeper, Schönborn, and others.

Of late, French writers especially have laid most stress on the cellular content of the cerebro-spinal fluid in the various affections of the nervous system, and particularly in meningitis, though after Lichtheim and Krönig had already pointed out the importance of this investigation. Widal and Sicard have originated the method of *cytodiagnosis*, and their teaching has been the stimulus, especially in France, to the production of such a number of investigations that they can no longer be followed. It may be asserted, however, that these works dealing with cytodiagnosis take up the most extensive place in contemporary French literature.

The formula of Widal referring to serous cavity effusions in general can be applied to inflammatory affections of the meninges. Thus *tubercular meningitis* is characterised by the *predominance of lymphocytes, purulent and cerebro-spinal meningitis* of *polynuclear cells.*

This doctrine has proved to be on the whole correct (observations of Ravaut, Achard-Loeper, Souques, Griffon, Faisans, Rocaz, Méry, Guinon-Simon, Nobécourt-Roger, Descos, E. Bendix, Milian, Laignel-Lavastine, Karwacki, Réné, Pinault, Concetti, Devaus, Séglas, Lair, and others). In individual cases, however, some variations from Widal's rule have been found. Thus Léri, Lewkowicz (*Presse méd.*, 1901), and others have in contrast to Widal found lymphocytes in the cerebro-spinal fluid of healthy people, in small or even in large numbers; Krönig had already recorded the presence of lymphocytosis in serous meningitis; in the observations of Rendu, Dufour, Méry-Babonneix (*R. n.*, 1902), in spite of a more or less marked lymphocytosis, the meningitis was not tubercular in character, and, conversely, polynuclear leucocytes may occur in tubercular meningitis (Lewkowicz, Brion, Léri). There is a certain dependence of the features on the stage and mode of evolution of the disease. Lymphocytosis (apart altogether from syphilis, tabes, and general paralysis) has occasionally been observed in typhoid fever (Widal, Vaquez, Variot, Méry-Babonneix), and also in herpes zoster. Finally, Widal himself has suggested that any meningeal irritation may lead to a lymphocytosis.

Schwarz and Bronstein ascribe only a limited value to cytodiagnosis (in the sense of Widal) in the recognition and differentiation of the various forms of meningitis, but the experience of the last few years has shown that the data of Widal and Sicard are essentially correct (see literature in the compilation of Raubitschek). Quincke lays most stress on investigation of the cells and organisms, and draws no important conclusions so far from the chemical or physical characters.

The following facts regarding the *technique* and methods of the investigation should be stated: The fluid drawn off by sterile methods (about 3-4 c.cm.) should be centrifuged in a test-tube for

at least 10 minutes, the contents poured out, and only the deposit adherent to the sides taken from the inverted glass by means of a pipette or glass rod. This is spread in a thin layer on a slide, fixed or dried and stained (with eosin, methylene blue, triacid stain, etc.). Widal and Sicard have described their method in the *Revue neurologique*, 1903, No. 6. According to Sicard and Schönborn from 2-4 lymphocytes per field with a magnification of 150-400 is within the norm; if the field is studded with leucocytes the condition is unquestionably pathological. This has been confirmed by Nissl, Meyer (*B. k. W.*, 1904, and *A. f. P.*, Bd. xlii.), Siemerling (*B. k. W.*, 1904), and others. Nissl counsels prudence in drawing conclusions from small quantities since the results are dependent also on the technique employed. The procedure employed by Nissl—who, for example, centrifuges for three-quarters of an hour—is described in the *Zentralbl. für Nervenheilk.*, 1904.

The following disturbances and ill-effects from the operation must be referred to: The needle may enter the sub-dural space, between dura and bone; it may be choked by fibrin-clot, or may snap, etc. The lowering of pressure caused by drawing-off of fluid may accentuate the symptoms, especially the headache, etc., or may cause new ones, such as vertigo and disturbances of equilibrium (Gumprecht). These are the more likely to appear, the greater the quantity drawn off and the more rapidly it is done. If a root of the cauda equina is touched, this may be manifested by pain in the corresponding nerve area. Henneberg has seen severe hæmorrhage follow lumbar puncture from injury to a vein. Unfavourable secondary effects have also been recorded by Voss (*D. m. W.*, 1903), from Passow's clinic. It is not impossible, although it has not been proved to occur, that a *circumscribed* purulent meningitis may be transformed by puncture into a *universal* one, and that a cerebral abscess may be caused to burst (Stadelmann, Oppenheim,[1] Heine). The operation has been stated to be without danger by many physicians who have seen no fatal results amongst the hundreds of cases in which they have performed it, as by Krönig, E. Meyer, etc., but the fact remains that death has occasionally followed almost directly on puncture, especially in tumours of the cerebrum or cerebellum (Fürbringer, Gumprecht,[2] Lichtheim, Rieken, Wilms, Leo Müller,[3] Masing [4]), and also in meningitis (Grunert,[5] Stadelmann, Strom Bull), aneurism (Krönig), cysticercosis (Krönig) and, uræmia. Gumprecht has collected from the literature fifteen cases of death from lumbar puncture and has added two personal observations to the list. Maystre [6] has dealt very fully with the question. Ossipow [7] working experimentally under my direction, has shown that in animals lumbar puncture may lead to hæmorrhage into the substance of the central nervous system, and L. Müller has seen the same result in a patient of Nonne's suffering from cerebral tumour; similar conclusions seem to follow from the observations of Finkelnburg, etc.[8] Ponjick has seen fatal intradural hæmorrhage following lumbar puncture (50 c.cm.) in a hydrocephalic child. Schönborn also has seen a hæmorrhage which he attributed to the loosening of a thrombus, and once, results which led to a fatal issue in a case of general paralysis.

For an account of the effect of puncture on healthy persons, we are indebted particularly to Nissl. Headache, vomiting, apathy, and inability for work practically always set in some five to twelve hours after the operation, and these last for 1-8 days; in one case there were psychical disturbances. In 48 out of 112 cases of lumbar puncture in

[1] *B. k. W.*, 1896 and 1897. [2] *D. m. W.*, 1900. [3] *Mitt. aus d. Hamb. Staatskr.*
[4] *Petersb. med. W.*, 1904. [5] *M. m. W.*, 1905. [6] *Thèse de Montpellier*, 1903.
[7] *Z. f. N.*, xix. [8] See on this question also Milian, *Semaine méd.*, 1902.

mental cases, symptoms occurred which lasted one to two or even ten days.

The procedure is by no means without risk, as has been emphasised by Nissl, who is so careful in his technique, and by Schönborn, whose results closely correspond with his, and it is hard to understand that nothing is said upon this point by French writers. Further conclusions as to the diagnosis can only be drawn from it with reserve. It should therefore not be employed indiscriminately, but only in those cases of suspected meningitis where the diagnosis is doubtful and its establishment necessary in the interest of the patient.

This position which I hold is also maintained by Münzer, Heine, L. Müller, and others. Quincke himself chiefly refers to the risk in its connection with cerebral tumours and recent hæmorrhages. With regard to the *therapeutic* value of lumbar puncture, which, according to the almost unanimous opinion of those who have had experience in this matter, is so far a limited one, and is only of real value in epidemic cerebro-spinal meningitis, see below, and in particular the chapter on hydrocephalus and serous meningitis.

Treatment.—If there is any suspicion of a commencing meningitis, absolute confinement to bed is essential. The head should be raised and the neck kept free. The patient should be disturbed as little as possible by outside stimuli, as even light or noise may be very painful.

It is always advisable to empty the bowels by administering *calomel* or some other aperient. Experience teaches also that prolonged application of an ice-bag to the closely shorn head serves to allay the malady. In robust persons, local *blood-letting* by leeches applied to the region of the mastoid process is very suitable, but excessive blood-letting should be avoided. The severe headache and obstinate vomiting can be controlled by morphia, and wild delirium makes the use of this drug necessary.

Some physicians praise the results of inunction of tartarated antimony ointment into the shaven head, the suppuration being kept up; at all events the application of a blister to the back of the head is sometimes a palliative measure. Energetic interferences of this kind, however, are to be deprecated, as they distress the patient and are of doubtful value. The administration of diuretics and the use of pot. iod. and mercury have warm supporters, but their usefulness is questionable. Dasoenberg, Fischer, and others recommend inunction with Crédé's silver ointment (2-3 grm. per day, employed as in a course of inunction). Injection of collargol into the spinal canal has also been tried.

Cold douching of the head, while the patient is in a tepid bath, has been employed for combating stupor. In many cases hot baths from 38° to 40° C., for 3-4 hours, which are specially useful in the treatment of epidemic crebro-spinal meningitis (Netter, Sevestre, Rendu, Raymond), have been curative. But Leyden, Kraus, and others have spoken with great reserve on the value of this method, although I have often seen good result from it. Hot packs to the extremities and hot fomentations to the head have also been recommended.

Above all it is important to trace the source of the disease and to *clear out any accumulation of pus in the neighbourhood of the skull, and any extra-dural abscess that can be reached.* Paracentesis of the tympanic membrane, *trephining of the mastoid process*, or drainage of a cerebral abscess may be necessary. It is beyond a doubt that in many cases of

commencing meningitis the disease can be held in check in this manner (Macewen, Gradenigo, Kander, Manasse, Alexander, and others). It is in the power of aurists especially to prevent the development of meningitis in many cases and to relieve it successfully when on the point of development. Since the demonstration of a turbidity in the cerebro-spinal fluid, and even the presence of leucocytes and perhaps even of bacteria are no evidence of the existence of a diffuse purulent meningitis, they form no contra-indication to the operative treatment of the ear-disease and the opening of the dura mater. It is only when undoubted signs of a diffuse purulent meningitis are present that no result of treatment can be expected (Jansen, Heine), and even under those conditions recovery may follow in some cases. It is obvious that proper treatment of injuries to the skull contributes to the prophylaxis of meningitis. It is scarcely necessary to mention that infection must be guarded against in operations on the skull and vertebral column, and particularly in lumbar puncture and lumbar anæsthesia.

Great uncertainty still prevails with regard to the question of *operative* treatment in this disease. A few cases are known in which puncture of the ventricles (Beck) has been of use, others in which the process, spreading from the ear, has been cured by incision of the dura mater and evacuation of the exudate lying in the meninges (Macewen, Lucae, Bertelsmann, Hammerschlag, Witzel, Gerber[1]). As a rule, in the cases cured in this manner, the condition has admittedly been a circumscribed meningitis in the region of the petrous temporal bone. Yet occasionally it has also been shown by lumbar puncture that a general meningitis was present. We are still, however, far from being able to draw up certain and definite indications for operative treatment. In recent years Haberer,[2] Friedrich,[3] Hinsberg,[4] Lermoyez-Bellin,[5] Manasse,[6] Kümmel,[7] Alexander,[8] Heine,[9] Ballance,[10] Körner, Radmann,[11] and others have made contributions to this question. Although their experience shows that a curative result may occasionally be brought about in the advanced stages of purulent meningitis by trephining, slitting of the dura, clearing out of accessible exudate and drainage, one must agree unconditionally with Jansen, Körner, and Heine, that any pus formed in the ear or skull should be evacuated on the first sign of meningitis. On the other hand, these results show that one should not shrink from taking operative measures even when there is marked development of meningitic symptoms. Körner is of opinion that one should desist from operative interference when there is definite proof of streptococci in the lumbar puncture fluid.

Lumbar puncture, which has been much employed in this connection (Lichtheim, Quincke, Heubner, Fürbringer, Chipault, and others), has had excellent results in no small number of cases of purulent meningitis, as in those reported on by Rieken, Jansen, Bormans, Hirsch, Langer, Netter, Nobécourt and du Pasquier, Mya, Donath, Tobler,[12] Kohte, Voss,[13] and others. Yet, as the authors themselves recognise, it remains doubtful whether lumbar puncture alone has been responsible for the cure in some of these cases. Alexander questions the efficacity of the measure,

[1] *D. m. W.*, 1904.
[2] *W. kl. W.*, 1903.
[3] *D. m. W.*, 1904.
[4] *D. m. W.*, 1904, and *Z. f. Ohr.*, l.
[5] *Presse méd.*, 1904.
[6] *Z. f. k. M.*, Bd. lv.
[7] *D. m. W.*, 1905, and *A. f. kl. Ch.*, Bd. lxxvii.
[8] *D. m. W.*, 1905.
[9] *B. k. W.*, 1906, and "Die Operationen am Ohr," Berlin, 1904.
[10] "Some Points in the Surgery of the Brain," London, 1907
[11] *Mitt aus Grenzgeb.*, 1907.
[12] *N. C.*, 1904.
[13] *Charité-Annalen*, xxix.

although he has seen undoubted improvement, but not recovery, follow its use. Nevertheless further investigations with this method are justifiable, especially in those cases where the signs of increased cerebral pressure become very marked. Puncture should then be repeated at intervals of one or more days, till the fluid becomes clear. The value, technique, and dangers of this method of treatment have been referred to above.

We have not as yet sufficient experience of the method recommended by Quincke of slitting the dura spinalis. *Dural infusion* with introduction of medicaments has not proved satisfactory. Laminectomy with incision of the dura and drainage (Paget) has only been tried once or twice. Rolleston and Allinghan, for example, have induced recovery (with persistent deafness) by this method in one case which they regarded as sporadic cerebro-spinal meningitis; also Bark, in a case in which the disease followed an injury to the vertebral canal.

We may sum up the recent experience in this by saying, that in purulent meningitis, which arises from a suppurative process in the cranium, surgical removal of this focus is the first essential, to be followed by operative relief of the meningeal pressure if spontaneous recovery is still delayed. Repeated lumbar puncture may have this result, but trephining and opening the dura mater, etc., seem to be a more certain method, although at the same time associated with greater danger.

Epidemic Cerebro-spinal Meningitis

For the literature, see Niemeyer, "Die epidemische Cerebrospinal Meningitis," etc., Berlin. 1865; Wunderlich, *Arch. der Heilk.*, vi.; Ziemssen-Hess, *A. f. kl. Med.*, 1865; Mannkopf, Braunschweig, 1866; Strümpell, *A. f. kl. Med.*, xxx.; Leichtenstern, *D. m. W.*, 1885; Heubner, "Eulenburgs Realenzyklop.," 1889; Schultze, Nothnagel's "Handbuch," *l.c.*; Jaeger, "Bibl. v. Coler," ix.; Weichselbaum, Kolle-Wassermann's "Handbuch," iii.; Weichselbaum, *W. m. W.*, 1906; Westenhöffer, *B. k. W.*, 1905 and 1906; Dow, *Med. Record*, 1906; Billings, *Med. Record*, 1905, Discuss.; Fowler, *R. of N.*, 1907; Cassel, *D. m. W.*, 1907; Lenhartz, *A. f. kl. M.*, Bd. lxxxiv.; Uhthoff, Graefe-Saemisch "Handbuch," 2nd Ed. Teil ii. Bd. xi. (copious literature, and not merely on opthalmological questions.

Purulent cerebro-spinal meningitis occurs not infrequently in epidemic form. In Germany this form has been familiar more especially since the sixties. It is undoubtedly an *infective disease*, due to micro-organisms. So far there have been recorded the pneumococcus of Fraenkel (Leyden, Jemma, Stadelmann), and more particularly the diplococcus intracellularis (Jaeger, Weichselbaum, Heubner, Fürbringer, Monti, Pfaundler, Mayer, Finkelstein, Osler, Cannet, Griffon, Lewkowicz, and others).

Most investigators now agree with the view of Fraenkel and Stadelmann, that this disease may be caused by Fraenkel's pneumococcus as well as by the diplococcus of Weichselbaum; Heubner regards the latter as the specific organism, and looks upon the pneumococcus only as causing sporadic meningitis.

Bettencourt-França (*Z. f. Hyg.*, Bd. xlvi.) and also Lingelsheim found Weichselbaum's diplococcus as a rule, but the latter, and with him Westenhöffer, lays stress on the frequency of mixed infection. This writer thinks it even possible that the real vehicle of infection is still unknown, and that the meningococcus only plays a secondary rôle. See also on this point Councilman (*Albany Med. Annals*, 1905), Kraus (*D. m. W.*, 1905), and for the behaviour of the meningococcus towards Gram's stain—Lingelsteim (*D. m. W.*, 1905), Dieudonné (*C. f. Bakt.*, Bd. lxi.), M'Donald (*R. of N.*, 1907).

Westenhöffer regards the raw pharynx as the point of entry of the causal organism; he found this region, especially the pharyngeal tonsil, always affected, and often also the ear, sphenoidal sinus, etc. This is confirmed by Goodwin. Children with a lymphatic constitution are said to be predisposed (Westenhöffer).

According to Flügge and Ostermann (*D. m. W.*, 1906) the infection is conveyed from affected individuals to healthy ones in their vicinity by means of nasal mucus containing cocci.

Epidemics occur usually in winter and spring, but they may also set in during the warm season. Direct transmission of the disease does not appear to take place. *Children* and *young persons* are most affected, in some epidemics children only. Some statistics show that 82 per cent. of the cases were children between one and ten years of age. In the most recent epidemic in Silesia children formed 79 per cent. of those affected (Kirchner). Persons over thirty are seldom seized by the disease.

There are *sporadic* cases, in which the clinical picture corresponds exactly with the epidemic form, and it can hardly be doubted that they are due to the same agent. This is confirmed by the fact that several individuals may be affected simultaneously in the same town, the same street, or the same house (Oppenheim, Sweet), although there cannot be said to be any epidemic. The poorer classes, and particularly persons living in crowded buildings, such as prisons, barracks, and workhouses are especially liable to be attacked. Recovery from one attack of the disease gives no immunity. Injuries may act as exciting causes, as shown, *e.g.* by a case of Michaeli's. In one instance the disease followed lumbar anæsthesia with stovain (Westenhöffer, *B. k. W.*, 1907).

The *clinical picture* is on the whole very variable, but in typical cases is somewhat as follows: The illness sets in suddenly or there may be preliminary symptoms, such as general malaise, shivering, restlessness, slight headache, and pain in the back and twinges of pain in the limbs, for some hours or even one to two days. Then the illness itself begins with *severe headache, vomiting, vertigo,* sensitiveness to light and sound, often also with *rigors*, less frequently and almost only in children, with *general convulsions.* The patient is restless and sleepless; the mind at first as a rule remains clear. At the onset, or after one to two days, the ominous *stiff-neck*, rigidity of the neck, the characteristic of the disease, appears.

The temperature is raised, although at first not usually over 39° C., but it shows considerable variations, as does the pulse, which is throughout rapid and irregular. Enlargement of the spleen is usually present, but it is not always demonstrable.

Within the next few days the headache increases and often becomes extremely severe, the fever persists or increases; the patient becomes actively *delirious*; the *rigidity* spreads to the *muscles of the jaw, back, trunk*, and *extremities.* The head is bent back almost to a right angle and trismus and opisthotonus set in. The abdomen is frequently retracted. Any touching of the skin or pressure on the muscles, and particularly any attempt at active or passive movement, elicits most acute pain, shown even during the delirium and the coma by painful contortions of the face.

The bowels are constipated; there is usually retention and later incontinence of urine. The amount of urine is frequently increased, and albumen and sugar are now and again present.

If the disease is taking an unfavourable course *coma* sets in, during

or towards the end of the first week, and paralytic phenomena become prominent. The face is pale and sharp featured, the pupils are usually dilated and *unresponsive to light*, ophthalmoscopy reveals a commencing *optic neuritis*; abnormal position of the eyeballs, squint, third nerve paralysis and facial paresis are not infrequently noted. If there is no coma or if the brain grows clearer *deafness* often now becomes noticeable. Conjunctivitis, chemosis, keratitis, and especially purulent iridochoroiditis may set in during the course of the meningitis.

The chemosis may be a result of inflammation of the orbital cellular tissue which Axenfeld has traced to the invasion by pneumococci. The keratitis may be neuroparalytic in character, *i.e.* a result of involvement of the trigeminus. Amaurosis is produced by direct involvement of the optic nerve (perineuritis descendens, optic neuritis); it may be present, however, with normal opthalmoscopic appearances and be the result of damage to the basal optic structures (chiasma or optic tract), or it may even be of cortical origin (Uhthoff, Depène, Oppenheim). Whether the optic neuritis is due to the micro-organisms themselves (Axenfeld has been able to trace them into the sheath of the optic nerve) or to the products of their metabolism is not settled. The purulent iridochoroiditis seems usually to be the result of mestastasis (Oeller, Axenfeld, Wintersteiner); it leads to amaurosis. Axenfeld characterises the condition as meningitis-ophthalmia. Panophthalmia with perforation rarely occurs. See further on this point Heine, *B. k. W.*, 1905; Uhthoff, *D. m. W.*, 1905, and "Bericht der 32. Versamml. d. ophth. Ges.," 1906; literature in Uhthoff, Graefe-Saemisch "Handbuch," 2nd Ed. Bd. xi.

While general or partial convulsions are not rare in the first stage and may occur repeatedly, the signs of a *monoplegia* or *hemiplegia* now show themselves, provided they are not obscured by the unconsciousness. Aphasia is seldom present. It is specially noteworthy, however, that in some cases the symptoms of a spinal paralysis also appear: *paraplegia*, *Westphal's sign*,[1] paralysis of the bladder, girdle pain, etc., which naturally are only distinctly recognised when consciousness begins to return during commencing recovery or remission. Paralysis of the cervical muscles sometimes occurs. Fowler lays stress on absence of the abdominal reflexes.

Skin-rashes, *erythema*, *urticaria*, *roseola*, *miliaria*, *purpura*, etc., are frequent and often appear early. But *herpes* is above all of diagnostic significance, and as a rule it appears within the first few days. The vesicles form chiefly on the lips, often also on the face, ears, and symmetrically over the extremities. Epidemics occur in which herpes is one of the symptoms of the disease in practically every case. They last usually only a few days and then dry up;[2] *micro-organisms* have been found in the vesicles.

Effusions into joints, of a simple or purulent nature, often take place.

In the cases about to terminate fatally coma persists, the patient wastes considerably, the urine and fæces are passed in the bed; there is profuse sweating, and meteorism may be present just before the end; the pulse is very small, rapid, and intermittent; the temperature rises to a considerable height (42° C. and over) or sinks far below the normal, and death follows usually about the end of the second or the beginning of the third week. In not a few cases, however, death may occur much later, after weeks or months, it may be because the disease in general had taken

[1] The tendon reflexes are usually exaggerated at first and may remain so in the later stages, but more frequently they disappear, though often only for a time. In general the examination is rendered difficult by the coma and raised temperature.

[2] Among recent papers on these points that of Einhorn, *W. kl. W.*, 1907, should be mentioned.

a prolonged course or because the end was the result of sequelæ such as bed-sore or exhaustion.

In the cases which run a favourable course the symptoms are as a rule less well marked from the outset; coma in particular is incomplete or altogether absent, and the temperature soon comes down or assumes a remittent character. In the course of the second week the troubles become milder, the uneasiness passes off; little by little the appetite returns, and the patient enters on convalescence. Unfortunately, however, he is still liable to *relapses*. Convalescence may run a long drawn-out course. A chronic condition, caused by chronic meningitis, hydrocephalus, etc., often follows, in which the irritative phenomena described persist with diminished intensity, and in particular paralytic symptoms, which will be mentioned subsequently, become prominent. Here also death may follow after a still longer period.

Koch has quite recently (*Therap. d. Geg.*, 1907) made important observations on the hydrocephalus which follows the disease.

The following *varieties* of this disease are of special interest: (1) The *fulminating* form: a healthy person is suddenly seized with a severe illness—chill, headache, vomiting, delirium, mania, or it may be coma, stiffness of the neck and paralysis, etc., develop in the course of a few hours, and death may ensue very quickly.

(2) The *abortive* form, mild and rudimentary, the very opposite to the preceding; these as a rule can only be accurately interpreted during the time of an epidemic. The patients complain of headache and pain in the back; they are somewhat restless and dazed; vomiting may set in, also local or general spasms, a slight degree of rigidity of the neck, etc. These symptoms may last from one or more days to one or two weeks and end in recovery. Frequently one has to deal only with a slight headache or pain in the neck along with general malaise; the relationship of such cases to the prevailing epidemic is doubtful.

(3) In a third category of cases the disease runs a protracted course, lasting many months. The fever also may assume an *intermittent* character, corresponding to a quotidian or tertian type. In the intervals the patient is without fever, and the other symptoms also diminish, to increase again with the fever. In some cases these intermissions only appear in the stage of convalescence.

Bertrand discusses this form in his thesis (Paris, 1904).

Among the abortive cases there occur also those in which the patient moves about (*formes ambulatoires* of the French, which have been described especially by Rendu, Sicard, Pinault, Léri, *R. n.*, 1902, and others); as a rule the disease is in these cases quite a mild one, yet this condition may be a severe one with an unfavourable prognosis. In some epidemics, the cerebral symptoms are insignificant as compared with the spinal ones. In connection with atypical forms see Comby-Netter (*Soc. d. Péd., R. n.*, 1906).

The recovery from epidemic cerebro-spinal meningitis is sometimes incomplete, certain symptoms remaining for a long time or permanently present. Amongst these are a tendency to *headache*, also *tinnitus aurium*, *strabismus*, *blindness* (from *secondary optic atrophy* or from *phthisis bulbi*, or very rarely from a lesion of the visual centres, according to the observations of Uhthoff, Axenfeld, Depène, and two cases which I have investigated), and, most common of all, *deafness*. The deafness sets in during the first

two or three weeks of the illness, more rarely during convalescence, and very seldom shows itself after some months have passed. If it occurs in early childhood, the result is that the child becomes deaf and dumb, and this condition, when acquired in childhood, is very frequently a residuum of epidemic cerebro-spinal meningitis. Even the abortive forms of this disease may have permanent deafness as a sequela. Usually there is a simultaneous disturbance of gait, which depends on defective co-ordination. Persons so affected walk unsteadily, tend to totter, and easily stagger and fall. This form of disturbance is specially marked in children; it tends to improve, however, in the course of months or years.

More rarely the meningitis leaves behind a hemiplegia, aphasia, or a paraplegia of spinal type, which persists for a long time or even permanently.

In one case which I have observed (*N. C.*, 1889) the following clinical symptoms remained after recovery from cerebro-spinal meningitis: occipital headache, giddiness, especially on bending back the head, difficulty in swallowing, nasal speech, left-sided facial paresis and contracture, paresis, contracture, and ataxy of the extremities on the right side, dulness of hearing, acceleration of the pulse, tendency to fall backwards on shutting the eyes, and lastly rhythmical twitchings of the soft palate and laryngeal muscles. I attributed these phenomena to a chronic meningitis of the posterior cerebral fossa and an encephalitic focus in the left half of the pons.

Weakness of intellect and memory may likewise be amongst the sequelæ of this disease. This question has been discussed by Sainton-Voisin (*L'Encéphale*, 1906).

Recently Netter, Chauffard, and Antony have dealt particularly with the sequelæ of meningitis; see also Bernard, (*Thèse de Paris*, 1903). Semerád (*Casop. lek.*, 1906) has seen persistent motor aphasia, due to the disease. Altmann discusses the prognosis (*Klin. Jahrb.*, xv). We cannot here enter into the relationship between cerebro-spinal meningitis and poliomyelitis anterior acuta (but see pp. 200 and 201).

Pathological Anatomy.—In fulminating cases, hyperæmia of the fine membranes and sero-fibrinous exudation may be the only change, or there may be no change at all. As a rule, however, there is a fibrino-purulent or quite purulent exudate in the meshes of the pia, on the convexity, and more particularly on the base of the brain, on the surfaces of the cerebellum, and in the spinal meninges over their whole extent, or more especially at the level of the lumbar enlargement. Over the spinal cord it extends most markedly on the posterior suface. Fowler lays special stress on the fact that the spinal meningitis may precede the cerebral. The cerebral ventricles contain a turbid, purulent fluid; on long duration a marked hydrocephalus may develop and the exudate in the meshes of the pia may undergo a caseous change (Ziemssen). Areas of inflammation and small abscesses may also permeate the substance of the brain (Klebs, Strümpell, Councilman).

The cranial nerves are partly embedded in and infiltrated by the exudate. The process may extend along the auditory nerve to the labyrinth and there set up a purulent inflammation; it is not improbable, however, that the infective agent has from the outset simultaneously involved the cerebral membranes and the inner ear. and that it may produce a bilateral hæmorrhage or purulent inflammation at this site. Otitis media with perforation of the drum is sometimes observed. Iridochoroiditis and panophthalmitis also occur.

Among the changes in other organs we must refer to broncho-pneumonia, pleurisy, and joint-affections.

For the pathological anatomy of the disease see Stroebe ("Handbuch d. path. Anat. d. Nerv.," Bd. i.) and Westenhöffer (*B. k. W.*, 1905 and 1906).

Differential Diagnosis.—The epidemic occurrence of the disease, the herpes, the early development of opisthotonus, and the spinal meningitic symptoms make this form of meningitis readily distinguishable from the others, especially from the tubercular form. Herpes seems to be a specially weighty factor in deciding the diagnosis as it is only observed in quite exceptional cases of tubercular meningitis. The presence of tuberculosis in other organs and the results of lumbar puncture (see above) should also be taken into consideration. Abortive forms can only be definitely recognised during the time of an epidemic, and lumbar puncture is simply invaluable as an aid to its diagnosis (Sicard, Netter, A. Schiff, Ferrand).

The pseudo-epidemic can be distinguished from the true form by bacteriological investigation, as the cerebro-spinal fluid contains either no bacteria or only streptococci and staphylococci (Baginsky, *B. k. W.*, 1907).

Typhoid is distinguished from epidemic meningitis by the characteristic behaviour of the temperature, the nature of the stools, meteorism, Widal's reaction, etc. Herpes is absent here,[1] and even if rigidity may occasionally be present in the neck, it never extends to the muscles of the trunk and extremities. Finally, the result of lumbar puncture is here also of the greatest differential value, as the cerebro-spinal fluid in typhoid is not turbid and contains no cellular elements or merely lymphocytes.

The only feature common to both *tetanus* and meningitis is the muscular rigidity, so that, as a rule, the diagnosis presents no difficulty.

Uræmic coma may give rise to many of the symptoms of meningitis (convulsions, vomiting, even rigidity of the neck, etc.), but the fever, herpes, hyperæsthesia, etc., are absent. Examination of the urine is not always conclusive, as albumen and casts are sometimes found also in epidemic meningitis.

Lépine (*Sem. méd*, 1906) discusses the question of the so-called uræmic meningitis.

In infants meningitis may also be confused with alcoholism (Ausset).

With regard to posterior basal meningitis of children, see the appendix (chronic meningitis).

Serous meningitis can only be mistaken for the milder forms of the epidemic disease. The temperature is usually normal or but slightly raised. The rigidity of the neck is much less well marked, and the contracture noticeably does not extend to the remaining muscles of the trunk. Herpes does not occur, although Henschen reports having observed it in one case. In doubtful cases the result of lumbar puncture may be decisive.

When epidemic meningitis is accompanied by swelling of the joints, it may simulate the picture of an acute articular rheumatism, but a consideration of the whole symptomatology and course will soon decide this point.

In hysterical stiff-neck, which is not infrequently observed during an epidemic, a rise of temperature and the signs of cranial nerve palsies are usually absent (see however, p. 761); on the other hand hysterical

[1] H. Schlesinger has recently (*W. kl. W.*, 1907) reported the occurrence of herpes in an epidemic of typhoid in Vienna.

symptoms are present, and it is almost always possible to establish the psychogenic nature of the opisthotonus.

With regard to the information obtained by lumbar puncture, see the previous chapter. As a rule there is present a turbid, purulent fluid which contains bacteria (especially the meningococcus intracellularis, rarely the pneumococcus, also staphylococci and streptococci) and many cellular elements, but especially *polymorphonuclear leucocytes*. It has already been insisted on, however, that any of these criteria may be absent. A certain dependence of the character of the fluid on the stage of the disease has been reported by Stadelmann, Councilman, Mallory-Wright, Labré and Castaigne, Griffon and Gaudy, and D. Gerhardt, a positive finding being most to be expected in the acute stage. Finkelstein, however, has found micro-organisms still present in the last stages.

The meningococcus may also be obtained and cultivated from the blood (Martini-Rohde, *B. k. W.*, 1905). With regard to the question of opsonins in the diagnosis and treatment of epidemic cerebro-spinal meningitis, only very few observations, such as those of Alice Taylor (*Lancet*, 1907) and Howston (*Brit. Med. Journ.*, 1907) have been published.

Prognosis.—This is always very grave, life being much endangered. The mortality of different epidemics varies from 20 per cent. to 80 per cent., amounting on an average to 40 per cent. or 50 per cent. In the last Silesian epidemic, of 3102 persons affected, 1789 died. Milder cases predominate towards the end of an epidemic. The chances of recovery are very small when the course is rapid, the fever high, and the coma early. But it is shown, *e.g.* by an observation of Stadelmann's, that even in severe cases recovery may ensue. On the other hand life may be endangered by hydrocephalus, even after a favourable termination of the meningitis (Zuppinger). The prognosis is more favourable in those cases which run a mild course from the start, especially if coma only appears late, is incomplete, or absent altogether. If a tendency to improvement does not appear during the second or third week, recovery is improbable. The prognosis is the more gloomy in that meningitis is fairly often associated with other infective diseases, especially with croupous pneumonia, more rarely with scarlatina, endocarditis, pericarditis, etc.

As regards sequelæ, blindness and deafness have on the whole a bad prognosis. If the latter has not been recovered from during the first three months after the outbreak of the disease, then it will in all probability be permanent, although I have myself once seen recovery follow after a longer time. The blindness of basal meningitis may be recovered from (Axenfeld, Oppenheim). Hydrocephalus may persist after recovery from the meningitis.

The so-called posterior basal meningitis, which has been described especially as a disease of childhood by British and American physicians, seems to be simply a combination of hydrocephalus and epidemic meningitis, or an hydrocephalus resulting from the meningitis (comp. Appendix, Chronic Meningitis).

Treatment

With regard to *prophylaxis*, see the proposals of Jehle (*M. m. W.*, 1906), Flügge (*D. m. W.*, 1907), and Krohmann (*Klin. Jahrb.*, 1907). The question of isolation requires no discussion.

The instructions laid down for the other forms of meningitis apply for the most part also to cerebro-spinal fever. The use of ice-bags,

local blood-letting, and aperients are advisable measures. Potassium iodide, sodium salicylate and digitalis have been employed without marked result. Ruhemann and Edlefsen recommend sodium iodide (2 per cent. solution in tablespoonful doses, or subcutaneously); Dazio advises subcutaneous sublimate injections,and Leszynsky subcutaneous injections of ergotin.

More recently treatment by hot baths has been greatly advocated by Netter, Heubner, and Raymond. I have also seen good result from this treatment, but Leyden and Kraus object to it, at least for adults. Göppert also is sceptical as to its value.

Leszynsky (*Med. Record*, 1906) employs warm rectal saline injections, and Hecht (*Therap. Monatshefte*, 1905) subcutaneous saline infusions.

In bad cases morphia can hardly be dispensed with; it is a powerful means of combating the pain, muscular rigidity, restlessness, and hyperæsthesia.

For surgical measures see the previous chapter.

In the last decade *lumbar puncture* has come more and more into favour as a curative measure (Ziemssen, Goldscheider, Wilms, Schiff, Netter, Kohts, Mya, Raymond,[1] Pellagot,[2] Tobler,[3] Auché, Lenhartz,[4] Drigalski, Finkelnburg, Bòkay, Zupnik[5]). These recommend that 70-90 c.cm. should be drawn off daily. Quincke is also in favour of frequently repeated puncture.

Göppert[6] and Koch do not ascribe any definite value to it.

The combination of hot baths and lumbar puncture has done well with me once or twice.

Trephining with opening of the dura and drainage of the meninges or ventricles has also been suggested for the epidemic form of meningitis, as *e.g.* by Westenhöffer, who expects satisfactory results from drainage of the inferior horn of the lateral ventricle with washing-out through a counter-opening beside the ligamentum atlanto-occipitale (?).

The attempts to produce and turn to account a meningococcal serum which have been made recently by Bonhoff, Lépine, Wassermann-Kolle,[7] Jochmann,[8] Flexner,[9] Schöne,[10] Stechow,[11] Schmidt, Rundle-Williams,[12] Hellmer,[13] Levy,[14] Schultz,[15] and others, seem to be more promising.

Wassermann recommends a single injection of 10 c.cm. of his serum for immunising, but for treatment in adults repeated doses of about 20 c.cm. are necessary; he regards the method as without risk, but only looks for good results when it is used early. Jochmann employs a serum got from Merck which should be injected during the first few days of the disease in doses of 20 c.cm. and over, either subcutaneously or into the spinal canal, after previous withdrawal of a corresponding amount of the cerebro-spinal fluid. He speaks favourably of its efficacy, and so also do Schöne and Neisser. By this treatment the mortality is said to have been diminished to 27 per cent. E. Levy thinks that for children 20 c.cm.

[1] *R. n.*, 1902. [2] *Thèse de Paris*, 1902.

[3] *N. C.*, 1904; *Schweiz. Korresp.*, 1905. This author does not hesitate to draw off large quantities—up to 100 c.cm., and in chronic hydrocephalus up to 650 c.cm. He finds this treatment specially valuable for postmeningitic hydrocephalus.

[4] *M. m. W.*, 1905; *A. f. kl. m.*, Bd. lxxxiv.

[5] *D. m. W.*, 1906. [6] *Klin. Jahrb.*, 1906.

[7] *D. m. W.*, 1906 and 1907. [8] *D. m. W.*, 1906 [9] *Journ. of Amer. Med. Assoc.*, 1906.

[10] "Therapie der Geg.," 1907. [11] "D. milit. Zeit.," 1907. [12] *Lancet*, 1907.

[13] *B. k. W.*, 1907. [14] *D. m. W.*, 1908. [15] *B. k. W.*, 1907.

for adults 30-40 c.cm. are necessary. Elder and Ievers [1] employed Rienzi's anti-pneumococcic serum. We have no remedy for post-meningitic deafness; diaphoretic measures are recommended.

Tubercular Meningitis [2]

This depends upon the invasion of the cerebral membranes by tubercle bacilli and tubercular poison; its significance is always that of a *secondary* infection. The primary tuberculosis may have its seat in the lungs, in the pleura, in caseous, bronchial, mediastinal, or mesenteric glands, in bones or joints, in the testicles or other part of the urogenital tract. Although no tuberculous forms may be found at the sectio, they are probably hidden somewhere; caries of the inner ear or of the sphenoid bone, or a caseous lymphhatic gland may specially be overlooked, but the primary tuberculosis may also have healed.

It is not known for certain by what route the poison reaches the cerebral membranes. Without doubt it may find its way into the blood-vessels and be carried to the brain with the arterial blood. Caseous material, for instance, may pass directly from the pulmonary veins. The spread of meningeal tubercle within the area of a meningeal vessel may also point to an *embolic* origin. Strümpell thinks that the poison spreads by the lymph-sheath of the nerves to the arachnoid sac of the spinal cord, and from there to the base of the brain. Leube is inclined to admit this mode of origin for some cases. Peron, on the ground of his investigations, supposes that the extension and dissemination of the process is carried out by the cerebro-spinal fluid, and recent experience acquired by lumbar puncture thoroughly supports this view. The conditions under which the infective material selects the meninges as the site upon which to settle and multiply are not easy to recognise. The child's brain is certainly predisposed to this condition, for tubercular meningitis appears by far the most frequently in *childhood* between the ages of 2 and 14, much more rarely in infancy. Persons between 15 and 35 are still liable to be affected, but after 40 it is very rare. It is possible that the active vital processes of the youthful brain, which we regard as associated with vigorous metabolism and therefore with increased conveyance of nutritive material, may render it so markedly susceptible to the disease.

In addition to this predisposition, *trauma*, mental over-exertion, and above all an *acute infective disease* may serve to bring on the illness. Thus a surprising number of tubercular children fall victims to tubercular meningitis after an attack of measles or of whooping-cough. A kind of epidemic occurrence of cases of tubercular meningitis has been referred to (Pott, Grawitz, Heubner, and others). Alcoholism must be regarded as a predisposing factor (Boix).

Pathological Anatomy.—The eruption of miliary tubercles in the fine cerebral membranes, and inflammatory changes in them, form the pathological groundwork of this disease. The sero-fibrinous, gelatinous, but rarely purulent exudate extends chiefly over the *base of the brain* in the region of the chiasma, between this and the cerebral peduncles; from

[1] *R. of N.*, 1907.

[2] The literature of this disease is so extensive and scattered that any attempt to cite it here must be given up; a very fertile source of references is the discussion of Uhthoff (*l.c.*).

this point it extends into the Sylvian fossæ, and stretches backwards to the under surface of the medulla, cerebellum, etc., and in the majority of instances it continues into the spinal cord membranes. The convexity of the brain is usually little affected, but there is sometimes turbidity and infiltration of the pia here also, especially over the sulci and along the vessels which are accompanied by filamentous streaks of the exudate. It is more rare that foci of tubercular inflammation with caseation form a layer of any considerable extent at this site.

The *miliary tubercles* which are embedded in the exudate, but which also occur in parts which are free from inflammation, are fine, grey-white, shining little nodules. They are sometimes few in number, sometimes very numerous, and, like the exudate, are to be found chiefly at the base in the neighbourhood of the vessels. They may be recognised most clearly when the pia is detached and held obliquely against the light. Isolated tubercles or even groups of them may also be seen on the dura mater in the region of the middle meningeal artery and its twigs. The choroid plexus is likewise usually studded with tubercles. The lateral ventricles contain a large quantity of serous, turbid, often also sanguineous fluid.

The degree of internal hydrocephalus may be considerable. The cerebral nerves or their sheaths are reddened, covered with exudate, and sometimes swollen. The brain substance is always altered. It is markedly injected, here and there adherent to the membranes, and in many places is in a condition of red softening. Tubercle eruption and local caseation extend in places into the cortex, and may even penetrate deep into the brain substance. A slight diffuse or disseminated *encephalitis* of the superficial layers of the cortex is practically always present. Nonne has recorded in one case the occurrence of a very extensive hæmorrhagic encephalitis (*Z. f. N.*, xviii.). Sometimes there are small hæmorrhages. It is specially noteworthy that areas of softening also occur deep in the substance of the basal ganglia, and may be traced to a tubercular arteritis with obliteration (rarely to compression of a vessel by the exudate).

Symptomatology.—The disease seldom attacks strong flourishing individuals. Generally they are weakly, pale-looking children or adult consumptives who are in a state of poor nutrition. The primary tuberculosis may have apparently died out or may be latent, or it may be only developing when the cerebral disease supervenes. The onset as a rule is preceded by *prodromal symptoms*, which are best marked in children, especially in those who have previously passed as sound. They are out of sorts, have no pleasure in their games, sit about brooding and with a downcast air, and are sometimes apathetic, at other times irritable, morose, and not infrequently for a time bewildered. Their sleep is restless and disturbed by mild delirium, or there may be *sleeplessness*. Soon *headache* sets in, at first slight and periodic, then severe and permanent, though with exacerbations. The child often complains less of headache than of pain in the abdomen and chest. Cerebral *vomiting* may come on in this prodromal stage and also a temporary increase in the body temperature. Sometimes the relatives are first of all struck by the loss of appetite, constipation, and emaciation which may for a long time precede the outbreak of the disease. After these symptoms have been present for a week or more, more rarely for a month, the features of *meningeal irritation* come prominently to the fore. The transition is usually

a gradual one ; sometimes, however, there is a sudden onset of severe *headache, delirium, stupor*, and *convulsions*—the signs of a severe brain disease. The delirium may be slight or severe, with great muscular unrest. Children, who in this stage, of course, are always bedridden, lie dozing uneasily, talking to themselves, grimacing, sometimes crying out loud, and throwing themselves about or trying to get out of bed. Adults in this stage are still sometimes able to go about ; they seem to be absent-minded, or are in a condition of dream-like, agitated confusion ; more frequently, however, the delirium here also is an *agitated* one ; it may correspond to alcoholic delirium in practically every trait. Wieg (*N. C.*, 1904) has recently described psychical states of this and other kinds.

The delirium soon develops into *somnolence*, or both conditions may be present from the outset, alternating or gliding insensibly the one into the other.

Whenever the sensorium clears up the patient complains of headache, often also of *vertigo*, and sensitiveness to light and sound. The headache may even break through the delirium, and it is shown by the patient grasping his head, groaning all the time. Children often wake out of their sleep or state of stupefaction with a piercing cry (hydrocephalic cry). They grind their teeth terribly—which healthy or nervous children also often do—and bore the head into the pillows. With the advance of the disease vomiting becomes more frequent and the stools are obstinately constipated. The urine, which may be retained or passed involuntarily, is small in amount and sometimes contains a little albumin.

Signs of *motor irritation* of various kinds make their appearance either at the very onset or later on. *Muscular rigidity* extends over the muscles of the back, trunk, and extremities. It consists in rigidity or stiff contracture, and is either general or limited in its distribution to distinct areas. A transitory tonic contraction also occurs in the muscles of the face, of one arm, or one side of the body. The jaw muscles may also be in a state of tetanic contraction. The knee-jerks are exaggerated, but it may not be possible to elicit them owing to the marked degree of contracture of the legs. Westphal's sign occurs practically only in the later stages ; inequality of the deep reflexes is not infrequently to be observed. *Fleeting muscular twitchings* become noticeable quite early, appearing now in this part, now in that, but especially in the facial region. General or local *convulsions* occur in this stage. The illness may even be ushered in with an *epileptic attack*. Frequently the convulsions affect one half of the body, corresponding exactly to the picture of a cortical epilepsy, and at the close of such an attack indefinite flickering twitchings continue to occur for hours. A *tetanic contraction* of the muscles may also be a transient but recurrent symptom. In isolated cases (Boinet,[1] Boncarut) the disease has been ushered in with choreo-athetotic movements in one half of the body.

The *temperature* is practically always raised, but remains on an average between 38° and 39° C., occasionally rising for a time considerably higher or sinking far below the normal. This irregularity and rapid rise and fall, even in the course of a single day, have something very characteristic about them. The pulse is as a rule slow, often arhythmic, and changes in its rate and character with extraordinary rapidity ; in the last stage of the disease it becomes very quick and small. The respiration is moderately

[1] *Gaz. des hôp.*, 1899.

accelerated; severe disturbances (Cheyne-Stokes breathing and allied types, stertorous or stridor-like breathing) usually set in only towards the end. The skin is dry; trophic disturbances, *e.g.* decubitus in typical and also in unusual sites, such as the ear, develop later on in the disease.

Of great diagnostic importance are the symptoms pointing to an *affection of the cerebral nerves*, which, on account of the prevailing basal spread of the process, practically always occur, although they usually become apparent only about the end of the first or in the course of the second week. Here, again, the *nerves to the eye-muscles* are specially involved, and consequently there is ptosis on one or both sides, contraction, dilatation, or inequality of the pupils, loss of pupillary reaction, etc. Complete ophthalmoplegia may even develop in rare cases (Oddo and Olmer [1]). The eyeballs are turned to the side, or they may look up or down, or move hither and thither, one extreme position succeeding another. *Nystagmoid twitchings* and paralytic squints are common symptoms. In a great number of cases ophthalmoscopic investigation gives a positive result—*optic neuritis* or choked disc. *Choroidal tubercles* are sometimes recognisable, although less frequently than in miliary tuberculosis.

Of the other nerves the *facial* is the most frequently affected. In addition to twitchings and contracture already mentioned, paresis and even paralysis occur. Contracture and paralysis both may be present. Paresis of the facial nerve sometimes shows itself first of all by incomplete closure of the lids. A basal paralysis of the hypoglossus is a much rarer occurrence.

At the same time the muscles of the body also become paralysed. A *monoplegia* of an arm or a complete *hemiplegia* makes its appearance, and may extend to the opposite side. These paralytic conditions are sometimes of fleeting duration, perhaps only following on a convulsive attack and then clearing up again; frequently, however, they persist, and there are cases in which a hemiplegia, developing gradually as a rule, but sometimes intermittently, formed from the outset one of the striking symptoms (Landouzy, Jaccoud, and others). *Aphasia* is not unusual and, indeed, is often an early sign of the disease (Chantemesse,[2] Frankl-Hochwart, Zappert,[3] E. Schlesinger, and others).

The focal symptoms may be due to inflammatory œdema, to pressure from a local accumulation of exudate, to local meningo-encephalitis, or lastly, to a softening which is the result of an arteritis obliterans (investigations of Friedländer, Cornil, Chantemesse, Zappert, Faure, Lavastine, Cruchet, and others). A very unusual occurrence is that recorded by D'Astros (*Arch. de méd. des enfants*, 1900), viz., thrombosis of the basilar vein with softening of the cerebral peduncle.

Zappert points out in this connection that cortical focal symptoms of every kind may indeed appear in the first stage.

Disturbances of *sensibility* are, as may be understood, rarely to be noted, but hemianæsthesia has occasionally been found. As a rule, hyperæsthesia of the skin and muscles is much more evident, specially in the early stages of the disease. Attempts at passive movement generally cause pain.

It is unusual for a condition of paralysis to develop early in the legs, or, instead of being spastic, to be flaccid, a condition which, in a case described by Oddo and Olmer, had an ascending character. Under these conditions the plantar reflex may be of the Babinski type. Anæsthesia

[1] *R. n.*, 1901. [2] *Thèse de Paris*, 1884. [3] *Wien med. Presse*, 1901.

of spinal origin in the legs is only recorded in a few cases (Hensen [1] and others).

In the second, and more especially in the beginning of the third week of the illness the *coma* gets deeper and deeper; the patient cannot be roused at all, and lies in an unconscious state. The paralytic phenomena are intensified and more widespread. Vomiting ceases. The pupils are dilated and cease to react. The features are wasted, the emaciation being considerable.

It is remarkable that even at this stage *deceptive remissions* sometimes occur, which, to the layman, may give rise to hopes of recovery. But the relapse follows quickly. Finally, the rigidity disappears, giving way to a general flaccidity; the patient can no longer swallow, the breathing becomes irregular and takes on the Cheyne-Stokes character. The pulse, hitherto slow, becomes suddenly or gradually very rapid, and after an agonal rise of temperature, even to 41° C. and over, and a considerable fall (even to 34° C.), *death* follows.

Course and Prognosis.—The disease may run a very acute course, and lead to death in a few days. Generally it extends over two or three weeks; if the prodromal stage be included, the whole duration of the disease amounts to a few months. It usually runs a steadily progressive course, though there are not infrequently remissions. The division into an irritant and a paralytic stage can scarcely ever be sharply made. Nevertheless, in the first week the symptoms of irritation are the most prominent, while later the features of depression and paralysis predominate.

But there are, both as regards symptomatology and course, other *atypical* forms. This applies especially to the tubercular meningitis of adults (Jaccoud,[2] Boix). It may even run a latent course or be entirely masked by the symptoms of the primary disease (Wunderlich, Fraenkel). There may be no rise of temperature at all. I have in some cases—also once or twice in children—seen absence of stiff-neck. The intelligence may remain quite clear until the last stage. As already mentioned, this disease may present the picture of delirium tremens. It may also set in with the symptoms of a focal brain lesion—monoplegia, hemiplegia, aphasia, cortical epilepsy—which may persist uncomplicated even for some time (Rendu, Chantemesse, Weintraud,[3] Zappert,[4] E. Schlesinger,[5] Landois [6]); here one is dealing probably always with an originally local, tubercular meningo-encephalitis (see below), which only later becomes generalised. Tubercular meningitis may also commence in an almost apoplectiform manner (Nobécourt-Voisin [7] and others). Finally, forms occur, although rarely, which run a chronic course, and in which there may be quite long intermissions. I know one case of this kind, in which the disease, combined with hydrocephalus, has lasted with intermissions for years. Mermann has reported a similar case, and Cruchet [8] has described one of quite unusual character—in which, it is true, the tubercular process was limited to the area supplied by the vertebral and basilar arteries, where there was an intermission of two years. Carrière-Lhote [9] also describe remissions of unusual duration. The observation communicated by Anglade-Chocreaux,[10] although vague, deserves also to be referred to here.

[1] *Z. f. N.*, xxi. [2] *Semaine méd.*, 1901. [3] *Z. f. k. M.*, xxvi.
[4] *N. C.*, 1902; *W. med. P.*, 1901. [5] *A. f. Kind.*, Bd. xxxix.
[6] *D. m. W.*, 1907. See also Panitscho, *Thèse de Paris*, 1902.
[7] Ref. *N. C.*, 1903. [8] *R. n.*, 1902. [9] *Revue de Méd.*, 1905.
[10] *Arch. de Neurol.*, xv.

The *prognosis* is bad. Most authors question if recovery ever really occurs. But from the observations of Dujardin-Beaumetz, Rilliet-Barthez, Schwalbe, Politzer, Jansen,[1] and more especially from a case reported by Freyhan,[2] in which tubercle bacilli were demonstrated in the fluid obtained by lumbar puncture, it can no longer be doubted that the disease may end in recovery. Henkel has described a similar case.

In recent years Schäche, Thomalla, Rocaz, Barth, Sépet (*Marseille méd.*, 1902), Jirasek (*R. n.*, 1904), Riebold, Rumpel (*D. n. W.*, 1907), Galliard, Guinon, Siredey, and Arazino (*Rif. med.*, 1903), have reported observations of this kind. Even if serous meningitis and other affections have here and there been mistaken for this disease, there is in my opinion no longer room for doubt as to the possibility of recovery from tubercular meningitis.

Rocaz' case, however, in which after an interval of two years relapse and death ensued, shows what caution must be exercised in judging of this recovery. See also on this question Pagès (*Thèse de Montpellier*, 1903), Mermann (*Beitr. z. kl. Chir.*, Bd. xxxiv.), and Claisse-Abrami (*R. n.*, 1905).

Slight signs of meningeal irritation which disappear later on, may occur in the course of a tuberculosis. It is difficult to say whether one is dealing in such cases with a tubercular meningitis about to develop and arrested at the onset, or whether the *tubercular poison can directly cause cerebral symptoms of a transient nature.*

Armand-Delille (*Thèse de Paris*, 1903) deals with the question what rôle the toxins of the tubercle bacillus play in the symptomatology of tubercular meningitis. See also Laignel-Lavastine, *Revue de Méd*, 1906.

With regard to the *differential diagnosis*, the previous chapter should be referred to. It contains also an appreciation of the diagnostic importance of lumbar puncture; positive results of this (finding of tubercle bacilli in the fluid) are of decisive value. As a rule the fluid is clear, but it may be turbid, opalescent, and in rare cases purulent or even hæmorrhagic. It is usually under considerable pressure and coagulates easily. Failure to find tubercle bacilli by no means implies that there is no underlying tubercular meningitis.

The accounts of writers who have dealt with this question are widely divergent. On the whole, however, it appears that with improvements in the technique—thorough investigation of the clot obtained on long standing, or the sediment got by centifuging, character of cultures, maximal staining and decolouring, etc., etc.—tubercle bacilli are more and more commonly found. This is particularly shown by the communications from Heubner's clinic (Slawyk-Manicatide, Holzmann, Finkelstein), and from the observations of Schwarz, Bernheim-Moser, Pfaundler, Breuer, Orglmeister (*A. f. kl. M.*, Bd. lxxvi.), Koplik (*Journ. of Amer. Med. Assoc.*, 1907), and others.

Moreover, French authors especially lay great weight on the finding of mononuclear lymphoid cells (Widal, Sicard, Ravaut, Faisans, Souques-Quiserne, Variot, comp. p. 766). Pfaundler has given interesting reports on the nature of the cerebro-spinal fluid in the various stages of tubercular meningitis.

An observation of Nobécourt-Voisin's (*Rev. mens. d. m. de l'Enf.*, 1903) shows that a cerebellar tubercle extending to the meninges may also lead to lymphocytosis of the cerebro-spinal fluid.

It is practically always possible to avoid mistaking this disease for tetanus. In one case in which the illness came on with trismus, there were tuberculous foci at the foot of both central convolutions. Confusion with typhoid and other acute infective diseases is possible, especially in childhood. The characteristic features have been mentioned. In the prodromal stage the severe cerebral lesion is frequently mistaken for a

[1] *D. m. W.*, 1896. [2] *D. m. W.*, 1894.

harmless catarrh of the stomach, for anæmia, etc. It is equally wrong, however, and the mistake is very frequently made, to suspect a commencing tubercular meningitis on every complaint of a tubercular or scrofulous child, when its appearance and temper are both bad. Scrofulous children of tubercular parents are frequently *neurotic*, and on such a foundation there develop anomalies of temper, loss of appetite, wasting and rapid pulse to which no special significance should be attached.

Whether the new tuberculin reactions of the skin and the ophthalmo-reaction described by Pirquet, Calmette, Wolff-Eisner (*B. k. W.*, 1907), and others will have a decisive value in the diagnosis of tubercular meningitis remains for further experience to show; from published accounts it seems doubtful. On this question see Stadelmann (*D. m. W.*, 1908), Gaupp (ditto), Citron, Feer, Morr (*M. m. W.*, 1908), and others.

Tubercular meningitis is not always easily distinguished from *miliary tuberculosis*. Yet in the latter the *pulmonary* features are more prominent, and there is usually also from the very start marked increase in the frequency of respiration and pulse. Tubercular meningitis may, however, appear in the train of a miliary tuberculosis (see an observation by Hensen, *Z. f. N.*, xxi., and others).

Tubercular meningitis is specially distinguished from the other forms by the long prodromal stage, the less rapid course, and, on the whole, the inconsiderable rise in temperature, etc. In childhood this form is always to be thought of first of all, but here also serous effusions occur with relative frequency, even in tubercular individuals (Quincke,[1] Münzer,[2] Patel,[3] Riebold [4]). Some of the factors of importance in the differential diagnosis have already been cited, and the others will be understood from the chapter on serous meningitis. The presence of choroidal tubercles is very significant. When caries of the sphenoid is present a purulent or a tubercular meningitis may develop; in children the latter is more frequent (Henoch).

It may be particularly difficult to recognise the disease when it occurs in infants. Variot and also Weill-Berthier (*Lyon méd.*, 1905) deal with this question.

Finally, we must mention those forms of *localised tubercular meningo-encephalitis* in which—usually in the region of the convexity—a circumscribed meningeal tuberculosis with caseous tubercles encroaches upon the cortex and thus forms the sole change (cases by Seitz,[5] Chantemesse,[6] Combe,[7] Boinet, Comby,[8] and others). The cortical matter in the neighbourhood of the focus is, as a rule, softened. In such cases the clinical picture does not correspond to that of tubercular meningitis but to a focal disease. Thus I have found [9] in a man who suffered from hemichorea and psychical disturbances, in addition to a solitary tubercle in the optic thalamus, a tubercular meningo-encephalitis over the right frontal lobe. In another case the headache and right-sided convulsions were associated with an aphasia which persisted while the accompanying symptoms disappeared. The autopsy a year later revealed a local tubercular meningo-encephalitis over the speech centre. The French

[1] *Volkm. Samml.*, 1893, and *Z. f. N.*, ix.
[2] *Therap. med. W.*, 1899.
[3] *Gaz. hebd.*, 1902.
[4] *D. m. W.*, 1906.
[5] "Die Meningitis tubercul. d. Erwachs," Berlin, 1874.
[6] "Étude sur la meningite en plaque," *Thèse de Paris*, 1884.
[7] *Rev. méd. de la Suisse Rom.*, 1898.
[8] *Gaz. des hôp.*, 1898. See also Hirschberg, *A. f. kl. M.*, 1887.
[9] *Charité-Annalen*, 1886.

investigators mentioned, who have studied this form specially thoroughly, refer to the fact that the region of the Rolandic fissure is the favourite site of this affection, and that in consequence focal symptoms of the motor area—cortical epilepsy, monoplegia, possibly also aphasia and hemianæthesia—are most in evidence. They have only described cases in which the disease took a fatal course through combination with tuberculosis elsewhere. I have reported[1] cases under my personal observation in which children fell ill with the appearances of an affection in the region of the Rolandic fissure, or in the region of the central convolutions and the frontal lobes, and the clinical picture brought to mind that of a neoplasm as well as of a chronic encephalitis. In all these cases recovery followed.

I have put forward the hypothesis—based on these observations—that localised tubercular meningo-encephalitis may appear and heal up in this way. Anatomical proof of this is hard to obtain, however ; but it is noteworthy that this affection has at various times been treated by surgical measures, and that, with excision of the focus, recovery has ensued (Raymond-Chipault, Lunz [2]). Monnier is the latest to insist that this disease may run its course under the guise of a tumour. We are also indebted to A. Saenger [3] for observations of this kind.

When the process is localised to other situations, the symptomatology becomes correspondingly modified. Thus Nonne (*Mitt. aus d. Hamb. Staatskr.*, 1905) describes a bulbo-cerebellar form in one case, with the appearances of acute ataxy, opthalmoplegia, etc., from the predominating involvement of the corresponding area of the brain.

Türk (*A. f. kl. Med.*, Bd. xc.) has described a case in which a yeast infection of the meninges was mistaken for a tubercular meningitis ; investigation of the fluid obtained by lumbar puncture led to a correct diagnosis.

Treatment.—With regard to this the chapter on purulent meningitis should be referred to. The hopes which had been put in iodoform have not been realised. Prophylaxis of tubercular meningitis is practically identical with that of tuberculosis: every tubercular focus should be treated *secundum artem* ; above all surgical treatment is frequently indicated to remove the source from which the tubercular virus might reach the brain. Children with tubercular tendencies must be guarded against overmuch mental work. Besides those referred to, careful bringing up, and living in fresh, pure air, are the most efficacious measures of prophylaxis.

In one case it is alleged that recovery followed on the continued administration of very large doses of potassium iodide (8-40 grm. per day). Specific treatment appears to lead the way to recovery, especially in those cases regarded as being a localised tubercular meningo-encephalitis (Oppenheim, Saenger).

Szalai (*Budap. orvos.*, 1906) and others say that they have had good results from the application of Bier's congestive bandages.

Surgical treatment so far has had no noteworthy results to chronicle. Opening of the cerebral sub-arachnoid space from behind and drainage has been recommended and carried out (Morton, Parkin, Kümmel). The vertebral canal has also been opened and permanent drainage employed after lumbar puncture, but without result (Wynter, Sahli, Lenhartz).

[1] *B. k. W.*, 1901. [2] *D. m. W.*, 1900 and 1904.
[3] *M. m. W.*, 1903. See further on this point Madeleine, *Thèse de Paris*, 1902.

Puncture of the ventricle has been undertaken as a palliative measure (Bergmann, Keen, and others).

The least harmful of these measures is undoubtedly lumbar puncture, which has brought about temporary improvement in quite a number of cases. It was employed also in Freyhan's case, but that author himself is not inclined to attribute the recovery to this treatment. Kohts and E. Schlesinger have only obtained improvement with this procedure, while Riebold (*l.c.*) has seen recovery follow after puncture repeated twenty-four times. Some cases, however, have been aggravated by the use of this method (Denigès-Sabrazès, Wentworth).

Appendix. Chronic Meningitis

If we exclude the syphilitic forms, which are discussed in the section on brain syphilis, we must regard chronic meningitis as a rare affection. Meningitis running a chronic course from the very beginning occurs in *chronic alcoholism*. It is localised chiefly on the convexity of the brain and is characterised by opacity and thickening of the fine membranes, not as a rule very marked. The same changes are to be noted in the diffuse diseases of the cerebral cortex—especially in dementia paralytica and senilis, and in hereditary chorea.

Nothing certain can be said with regard to the *symptomatology*, as it is usually a case of an accidental post-mortem finding, or the symptoms dependent on the brain lesion are so prominent that those due to the chronic meningitis cannot be distinctly separated from them. In one case recently described by Raymond (ref. *R. n.*, 1906), the clinical picture was reminiscent of a general paralysis.

Chronic meningitis ossificans, which is sometimes found in mental diseases, in epilepsy, and occasionally in persons who have suffered for years from obstinate headache, is scarcely of any clinical interest; it may lead to the formation of an osseous envelope which rests on the cerebral cortex like a cap.

We need only mention here that sarcomatosis and carcinomatosis may spread after the fashion of a diffuse meningitis; the same applies to chronic cysticercus-meningitis (Askanasy,[1] Rosenblath,[2] Oppenheim).

Chronic basal meningitis is a better-known form of the disease. In the great majority of cases it is certainly of *syphilitic origin*. Sometimes it is the residue of a previous acute cerebro-spinal meningitis. Isolated cases have been observed, however, which point to the occurrence of a *simple primary chronic basal meningitis* (Huguenin,[3] Carr,[4] Carmichael,[5] Lunz [6] and others), though some of the cases so regarded have probably been either *serous meningitis* or a combination of serous meningitis with simple fibrous basal meningitis. Compare on this point Krause-Placzek (*B. k. W.*, 1907). It leads to opacity, thickening, and growing together of the membranes and the cortex; the cerebral nerves, especially the *optic*, are similarly involved. It is of real importance, however, in that the means of communication between the ventricles and the subarachnoid space become blocked, and the conditions necessary for the development of *internal hydrocephalus* are thus brought about. Some authors go so far as to attribute acquired (idiopathic) hydrocephalus always to a basal meningitis of this sort, which is incorrect (see chapter on hydrocephalus). It is impossible to draw a very characteristic clinical picture of this simple basal meningitis, owing to the scarcity of observations which have been confirmed by sectio. As a rule there was obstinate *headache*, with exacerbations from time to time, and *vomiting*: more rarely, a feeling of giddiness or general convulsions. Slight rise in temperature has also been recorded. In addition to this there is *optic neuritis*, leading to *atrophy*, sometimes also ocular paralyses, and in some cases paralysis of other cranial nerves. The disease runs a chronic course and may come to a standstill, or the added hydrocephalus may bring about a fatal termination.

In the early years of childhood there occurs a chronic meningitis, probably usually of a syphilitic nature, which is limited exclusively or mainly to the posterior cerebral fossa. The meninges are thick, like bark, adherent to one another and to the cerebellum, medulla oblongata, etc. The process shows itself in rigidity of the neck, vomiting, general convulsions of an epileptiform and

[1] *Ziegler's Beitr.*, vii. [2] *Z. f. N.*, xxii. [3] Ziemssen's "Handbuch," etc., xi.
[4] *Med.-Chir. Trans.*, 1897. [5] *Ed. Med. Journ.*, 1897. [6] *Journ. neur. Korsakow*, 1902.

tetanoid nature, and rigidity of the muscles of the trunk and limbs. Blindness is often present, and is the result of a secondary hydrocephalus, which presses the floor of the third ventricle against the optic chiasma. In two of my cases there was complete amaurosis, with conservation of the pupil reaction, and no ophthalmoscopic changes, so that I presumed there had been an extension of the process to the occipital lobes. Ocular paralyses and nystagmus have likewise been recorded. Further, the cerebral nerves arising in the posterior fossa may be affected and corresponding paralytic symptoms ensue. The prognosis is a serious one, the child usually dying of hydrocephalus. Arrest and improvement may occur, however. In such cases antisyphilitic treatment is always indicated.

On the other hand there is a more acute form of this disease, or at least a form with an acute onset, occurring in infants, which seems to be related to epidemic cerebro-spinal meningitis, as is shown particularly by bacteriological investigation. Cases of this kind have been described by Thursfield, Gee-Barlow (*St Barth. Hosp. Rep.*, 1878), Carr (*l.c.*), Still, and recently more especially by Koplik (*Amer. Journ. of Med. Sc.*, 1905). See also Hildesheim (*Brit. Med. Journ.*, 1906), Corkhill (*Brit. Med. Journ.*, 1906), Ballance, and Longmead (*Practitioner*, 1907). Thursfield hopes for good results from lumbar puncture.

Disorders of the Cerebral Circulation

Our knowledge of the disorders of the cerebral circulation and the phenomena dependent on these is decidedly incomplete—pathological anatomy failing us here entirely. The amount of blood in the brain as seen after death may bear no definite relation to that contained in it during life; it is dependent upon such factors as the manner of death, the position of the body, etc. Our knowledge, therefore, is founded upon the observation of cerebral symptoms in patients suffering from general anæmia or plethora, and of those produced by sudden loss of blood or by sudden interruption of the supply of blood to the brain. The symptoms produced experimentally by ligature of the vessels supplying the brain must be applied with great reserve to the study of the circulatory disturbances in the human brain. Kussmaul and Tenner have produced general convulsions and loss of consciousness by ligature of the carotid and vertebral arteries in animals. Mosso has confirmed this. Loss of consciousness has also been observed in man after compression of both carotids.

Cerebral Anæmia

Anæmia of the brain, in its acute form, is produced by sudden great loss of blood, by the flow of large quantities of blood to other organs or parts of the body, *e.g.* in paracentesis of the abdomen, in the sudden evacuation of a general ascites, in precipitate delivery, in the use of Junod's boot; further, by the obstruction of the blood-supply to the brain from acute cardiac weakness or spasm of the cerebral arteries.

Cerebral anæmia is usually, but not invariably accompanied by pallor of the face and of the mucous membranes.

If the *cerebral anæmia* be acute it leads to the following *symptoms*: the patient has a sense of darkness before the eyes and *disturbance of consciousness*: he feels as if the earth were rocking under his feet. In addition to these, *tinnitus aurium*, *nausea*, and sometimes *vomiting* are present; sensory perceptions are weakened; *apathy* and *lethargy* set in, the *pupils are contracted*; ultimately consciousness may be completely lost. In great loss of blood *unconsciousness* is, as a rule, complete, the reflexes are lost, the pupils are dilated, and general convulsions may set in. The symptoms obtained experimentally by Kussmaul and

Tenner correspond with those produced by Naunyn in patients with arterio-sclerosis by compression of the carotids, viz., unconsciousness, convulsions, dilatation of the pupils, and slowing of the pulse. Naunyn ascribed these to cerebral anæmia caused by the deficient amount of blood in the circle of Willis. The slighter degrees produce an attack of *syncope*; the patient is depressed, feels ill, and as if his senses were going; he partially loses consciousness, and there may be yawning, tinnitus aurium, and shimmering before the eyes. The skin becomes pallid and cold, and is sometimes covered with cold perspiration. The patient loses consciousness, but not, as a rule, so completely that strong sensory stimuli cannot be felt. The pulse is small, usually slightly accelerated, sometimes intermittent; the breathing is either slow or rapid, sighing, or irregular. Convulsions do not occur. The attack lasts, as a rule, for a few minutes, but may continue for an hour. Consciousness frequently returns as soon as the patient lies down and is lost again when he sits up. The cause of the faint attack is the sudden interruption of the supply of blood to the brain. This may be due to a general spasm of the cerebral arteries or to defective cardiac action, usually the latter. The attack may be brought on by great excitement, fear, anger, grief, unpleasant sensory impressions, *e.g.* the sight of blood, and by physical pain. Neurotic individuals are predisposed to attacks of faintness, and this tendency is increased by general anæmia. Heredity, too, plays a part, and the predisposition to such attacks may be transmitted through several generations.

I have had under treatment a young girl, who, like her mother, became faint every time her hands touched cold water. I know one family in which six members have more or less severe attacks of fainting during any mental excitement, one of whom is liable to an attack of deep unconsciousness under the influence of a certain idea.

Chronic anæmia of the brain, as in chlorosis, pernicious anæmia, leukæmia, and after repeated hæmorrhage, occasions *intracranial pressure, drowsiness, apathy, vertigo, tendency to faintness, tinnitus aurium, weakness of memory, sleeplessness*, and more rarely hallucinations. These symptoms are most marked when the patient is sitting upright and least when he is lying down. One of my patients, who had become very anæmic owing to previous loss of blood, was seized in the morning after rising by attacks consisting of vibrations before the eyes, followed by great giddiness, and finally by vomiting. The attack lasted from one to two hours but was shortened when the patient lay back. The *delirium of inanition* is also due in part to anæmia of the brain, although here deficient nutrition is the most significant factor. Obstinate diarrhœa, especially in children, may also result in symptoms of cerebral anæmia. The most severe forms of this nature are those described by Marshall Hall as *hydrocephaloid*. The cerebral symptoms that occasionally threaten in chlorosis, and even simulate those of a tumour, are caused chiefly by complications (sinus thrombosis, meningitis serosa ?).

The *prognosis* of cerebral anæmia is favourable in the less severe forms; the fainting attacks are hardly ever dangerous to life. If it can be ascertained that there exists an habitual tendency to fainting, there is always a prospect of cure, even where there is a profound degree of unconsciousness. In severe hæmorrhage, however, coma may set in and may have a fatal result. The onset of general convulsions is always an unfavourable sign; so is dilatation of the pupil and absence of the light

reflex. If contraction of the pupils and reaction to light return, the prognosis as to life is favourable, even after considerable hæmorrhage. If the danger to life be removed, the prognosis is still unfavourable in that visual disturbance and optic atrophy may be a permanent consequence of the loss of blood (*cf.* p. 722). Focal cerebral symptoms less often follow profuse bleeding: in one case (Bouveret's [1]) they were traced to œdema. The symptoms of cerebral paralysis which sometimes follow great loss of blood during delivery might, however, be due to arterial or venous thrombosis (Hösslin, see next chapter). According to more recent investigations it appears that in acute anæmia, fine changes develop in the nerve-cells of the brain as the result of the severe hæmorrhage (Scagliosi,[2] Soukhanoff [3]).

Treatment.—In acute cerebral anæmia the *horizontal position* must be immediately assumed, preferably with the head lowered. The blood-supply to the brain can be further increased by firmly bandaging the extremities from below upwards. If deficient cardiac action or faintness be the cause, then *stimulants* are required: wine, brandy, ether, camphor. *Stimuli to the skin, e.g.* sprinkling with cold water, mustard-plaster, faradisation, are also of use. *Nitrite of amyl* is of value in severe cases—a few drops may be put on a handkerchief and held under the patient's nose. In severe cases of cerebral anæmia *artificial respiration*, and finally transfusion, may be considered.

The treatment of persistent cerebral anæmia is that of anæmia in general.

Cerebral Hyperæmia

Cerebral hyperæmia may be *active* or *passive*. A persistent arterial plethora of the brain is just as problematical as is a general plethora.

In addition to the few cases of *plethora vera* reported by Weber-Watson, Hirschfeld, Hutchinson-Miller, and Glässner, Westenhöffer [4] has recently contributed an indisputable one, and, agreeing with Türk,[5] he has been able to refer the affection to a condition of the bone-marrow corresponding to that in early youth. Further, there are people of stout build and short neck, who, at all times or after a meal or any physical exertion, get red in the face, and in whom the symptoms which we are inclined to attribute to cerebral hyperæmia are constantly present or at least occur on the most trivial occasions. F. Müller refers to the increased blood-pressure and to the polycythæmia in this diathesis.

We have no certain grounds for assuming that continuous mental overstrain causes a permanent arterial hyperæmia of the brain.

We have, however, more exact knowledge regarding a transitory increase in the supply of arterial blood to the brain. It can scarcely be doubted that the condition here is one of so-called *congestion*. There are individuals who often suffer without any recognisable cause, or after excitement, over-indulgence in spirituous liquors, a substantial meal, or even after strenuous mental work (Bastian), etc., from attacks which are described as *determination of blood* to the head, *fluxion*, or active *congestion*.

They become suddenly hot, the blood mounts to the face and to the head; there is throbbing in the temples, neck, and head, shimmerings

[1] *Revue de Méd.*, 1899 [2] *D. m. W.*, 1898. [3] *Journ. de Neurol. et d'Hypnol.*, 1898.
[4] *D. m. W.*, 1907. [5] *W. kl. W.* 1907.

and scintillations before the eyes, followed by headache and vertigo and mental confusion. After a few minutes or in half an hour the slighter attacks have passed off. During the attack the face and neck are very congested, the conjunctivæ may be very much injected. The skin is very hot over the head and especially the ears, the pulse is slowed or accelerated, full and tense, the carotid and temporal arteries pulsate, and the pupils may be contracted.

The severe forms are accompanied by a greater disturbance in consciousness and even by loss of consciousness or mental confusion, or by acute maniacal delirium. Fever may also be present. Conditions of such a nature have been found in children and young people and have been regarded as active hyperæmia of the brain. The symptoms had reached such a degree that they simulated a very acute meningitis, whilst they completely disappeared in a few hours or in the course of a day. A. Katz [1] has published a fatal case—confirmed by autopsy—in which the *active idiopathic congestion* had developed after an injury. A paralytic form of arterial hyperæmia has also been described, viz., an apoplectic attack with transitory symptoms of paralysis (hemiplegia), or even a fatal termination without any corresponding pathological appearances in the brain. Yet it has not been proved that a simple hyperæmia can cause paralytic conditions. It is certainly a fact deserving attention that amyl-nitrite may produce a temporary aphasia, but here it may be due to a toxic influence. General and even unilateral convulsions may be the result of an active congestion of the brain.

Determination of blood to the head is probably to be attributed to an inherited or acquired *instability* of the vascular nervous system. This may act in such a way that relatively slight causes may produce a *dilatation of the cerebral vessels*, especially the capillaries, accompanied by increased activity of the heart. This may be one of the symptoms of neurasthenia and hysteria. Masturbation plays an important part in producing this condition.

The diminution of the blood-supply to other parts of the body must be recognised as a further cause of active cerebral hyperæmia, *e.g.* the sudden contraction of the cutaneous vessels during a cold bath may cause a flow of blood to the brain. An analogous effect may be produced by the suppression of regular hæmorrhages (menses, hæmorrhoids), and two cases under my observation (see next chapter) argue in favour of this possibility. Certain poisons which dilate the cerebral vessels cause increase in the blood-supply to the brain. To this group belong alcohol, nitro-glycerine, amyl-nitrite, etc.

The symptoms of *sunstroke* have been referred by some writers to cerebral hyperæmia, by others to thrombosis and multiple capillary hæmorrhages in the medulla oblongata (Silbermann, Herford, and others), but anæmia (Arndt), œdema of the pia (Jackson), meningitis, and finer cell changes, etc., have also been found or assumed to exist.

The condition is attributed by Maas and Senftleben (*B. k. W.*, 1907) to dehydration of the tissues and auto-intoxication. See also Hiller, "Der Hitzschlag auf Märschen," *Bibl. Coler*, xiv., and Goebel, "Über die Nachkrankh. d. Hitzschlags," *Inaug. Diss.* Berlin, 1905. Injection of water into the intestine or subcutaneously has therefore been used in treatment.

The clinical symptoms of so-called heat-apoplexy are likewise very varied: rise of tempera-

[1] *Arch. de Neurol.*, 1901.

ture, headache, psychical disturbances, loss of consciousness or deep coma with a considerable rise of temperature, paralytic conditions of paraplegic or hemiplegic character, disturbances of speech, ataxia, etc. (observations by Jackson, Cramer, Obersteiner, Kussmaul, Andrew-Duckworth, Hiller, Schwarz, Herford, Rothmann, and others). More recently observations have been published by Senftleben, Revenstorf (*D. m. W.*, 1907, and *Ärztl. Sachverst.*, 1907), Nonne (*D. m. W.*, 1907).

A great variety of diseases have obviously been included under this term (Noir).

Passive hyperæmia—as opposed to active—is chiefly a chronic condition due to compression of the veins carrying the venous blood from the brain—viz. the jugulars, by tumours in the neck or strumous glands, the superior vena cava by mediastinal tumours, etc., to uncompensated heart disease, or to emphysema of the lungs, etc. Coughing, straining, sneezing, stooping, tight collar, etc., may temporarily produce slight venous hyperæmia of the brain or increase an existing one. The symptoms are *drowsiness* or *insommia*, *intracranial pressure* increased by coughing, *vertigo*, *apathy*, *somnolence*, and *slight confusion*. These symptoms are usually, but not always, more marked when the patient is lying down, especially when the head is low. The condition of fear and confusion which arise on each attempt to fall asleep is very distressing. In severe cases stupor, coma, and even epileptiform attacks may set in.

The signs of passive congestion are usually evident on the face and mucous membranes.

The *prognosis* of simple congestion in the head is on the whole favourable. Danger arises only if organic disease of the vessels or of the heart is the cause.

It is also true that the " habitus apoplecticus," in the meaning described above, and particularly " plethora vera," increase the tendency to cerebral hæmorrhage.

In venous hyperæmia the prognosis depends on that of the primary disease : it is almost invariably an unfavourable one, yet treatment (excision of glands, etc.) may be effective.

Treatment.—To prevent cerebral congestion excesses of every kind must be forbidden, and strong coffee and tea, alcoholic liquors being permitted in only very small quantity, if at all. Mental and physical over-strain should be unconditionally forbidden. Hydropathic treatment, cold packs, lukewarm sitz-baths, baths in running water or in alternate changes of hot and cold water—are specially of value in combating the tendency to congestion.

During the attack the patient must maintain a sitting or lying posture or one with the head raised and the neck free from all clothing. The room must be cool and well-ventilated. Cold fomentations to the head and sinapisms on the neck, breast, and calves are worth trying in slight attacks. Faradisation of the skin of the trunk and especially of the feet may be of considerable effect. If the attack be severe or prolonged then in strong individuals it is justifiable to try blood-letting (venesection, cupping, leeches over the temples, Bier's aspirator), purgatives (calomel, stimulating enemata, etc., or in persistent hyperæmia the use of aperient waters), hot foot-baths, hot-packing of the lower extremities, etc. It is very doubtful if ergot can counteract hyperæmia of the brain.

Passive hyperæmia is successfully treated only by removal of the cause. Thus the extirpation of a tumour, of strumous glands, the

regulation of the heart's action by digitalis, etc., may be necessary. Symptomatic treatment consists in purging, and mitigating the headache and the sleeplessness by narcotics, but we must be careful in the use of opiates and chloral hydrate. Strychnine is said to be harmful in these conditions. Bromide preparations are to be recommended.

Cerebral Hæmorrhage

Literature: s. Monakow, Nothnagel's "Handbuch," etc., ix. 2. Aufl.

Hæmorrhage into the cerebral substance is one of the most frequent diseases of the brain. It may occur at any age, but the great majority of cases affect the *later* years of life; before forty this affection is rare and in childhood exceptional. Males are more frequently attacked than females.

The hæmorrhage is almost always the result of a *vascular disease.*

The most frequent disease of the arterial cerebral vessels is *atheroma* (*sclerosis of the arteries* according to Marchand). But whereas this attacks chiefly the larger vessels and is thus especially the origin of the brain softening by thrombosis, hæmorrhage on the other hand is brought about for the most part by a rupture of the intra-cerebral branches of the arteries of the circle of Willis, especially the artery of the Sylvian fissure. This is the seat of election of the *miliary aneurisms* (Charcot, Bouchard [1]), which may it is true be present along with arterio-sclerosis, but are by no means always combined with it. They rarely develop before the fortieth year and thereafter increase in frequency. The rupture of a miliary aneurism is usually found to be the cause of cerebral hæmorrhage, yet this is by no means a "conditio sine qua non." The frequency of this disease of the bloodvessel wall has without doubt been overrated, for some observers have interpreted the simple bulging of the lymphatic sheath by the effused blood as an aneurism (Egger, Stein [2]).

Other diseases of the cerebral vessels, *e.g.* hyaline degeneration (Weber), may also lead to rupture and hæmorrhage, and smaller hæmorrhages might quite well arise through diapedesis. The hæmorrhages caused by the rupture of aneurisms of the larger vessels are found mostly in the meninges, especially those at the base of the brain, where they remain unnoticed. The hæmorrhages that arise from blockage of the vessels play on the whole a subordinate part, but the local irritation of the vessel wall by embolus itself produces changes in it which bring about rupture. *Endocarditis verrucosa* may give rise to rupture, and cerebral hæmorrhage is a consequence of embolic material getting into the brain and producing locally a pathological condition of a vessel wall and the formation of an aneurism (Ponfick). In Simmond's [3] experience cerebral hæmorrhages of this origin are not infrequent in childhood, and a case of Irvin's also confirms this view. But rare cases of fatal cerebral hæmorrhage in childhood have been observed without any recognisable cause. Fat embolism of the cerebral vessels, which Ribbert thinks may result from comminuted fracture of bone, may also lead to cerebral hæmorrhage.

Cerebral hæmorrhage may also be brought about by rupture of veins and venous sinuses.

The causes of cerebral arterial disease are very numerous, Advanced years, or rather *senility*, is the most important and most frequent.

[1] "Arch. phys. norm. et path.", 1868. [2] *Z. f. N.*, vii. [3] *D. m. W.*, 1901.

Deterioration of the vessels is indeed the chief factor, and this may be brought about even in youth by excessive demands being made upon them.

Then follow *intoxications* and *infections*: thus *alcoholism* and *lead-poisoning* are not infrequently, and *syphilis* is very often a cause of disease of the cerebral arteries. These conditions thus play a not inconsiderable part in the etiology of cerebral hæmorrhage, although syphilitic affection of the vessels is far more frequently followed by softening than by hæmorrhage.

Probably excessive use of *nicotine* may injure the vessel wall. Whether an analogous effect can be ascribed to *caffeine* is not certain, but I have known cases in which an excessive amount of coffee—*e.g.* among coffee- and tea-tasters—produced arterio-sclerosis at an early age. Its experimental production by injections of adrenalin is very interesting (Josué, Erb, Lissauer, *B. k. W.*, 1905, Ziegler *Beiträge*, Bd. xxxviii., Falk, *D. m. W.*, 1907). Rotky describes *cerebral hæmorrhage* in acute phosphorus poisoning (*Prag. med. W.*, 1906).

Acute infective diseases favour the occurrence of cerebral hæmorrhage, yet it has not often been observed in such conditions. We assume that they induce arterio-sclerosis and other diseases of the vessel wall. Thus typhoid-hemiplegia (Eichhorst and others) may be traced occasionally to hæmorrhage but more frequently to thrombosis and other changes (Hawkins, Wallenberg, Osler). In all general diseases which depend on a *hæmorrhagic diathesis*, e.g. purpura, pernicious anæmia, and leukæmia, the brain is not infrequently the seat of hæmorrhages, usually multiple. *Gout* and *nephritis*, especially granular contracted kidney, likewise predispose to cerebral hæmorrhage. It is also frequently found in eclampsia. The cerebral paralysis appearing in *diabetes* is often attributable to auto-intoxication, but may also, as observed by Klippel and Jarvis,[1] be due to cerebral hæmorrhage.

Experience puts it beyond doubt that heredity is of some importance in the occurrence of cerebral hæmorrhage. The tendency to apoplectic seizures may descend through generations.

If the cerebral vessels be diseased then hæmorrhage may occur without any further recognisable cause, but its occurrence is promoted by all the factors which cause an increase of blood-pressure. Thus *hypertrophy of the left ventricle*, especially if uncompensated, is always a menace to the brain, and miliary aneurisms are frequently directly associated with this. It is improbable that healthy vessels can rupture under such a condition. The relation of cerebral hæmorrhage to kidney disease is due in part to the same condition: on the other hand the kidney disease most commonly associated with it—granular atrophy—is frequently connected with an arterio-sclerosis of the cerebral vessels.

The immediate cause of the cerebral hæmorrhage is frequently some temporary increase in the blood-pressure. Thus any severe muscular strain may occasion it, and this applies also to forced expiratory movements in coughing, sneezing, or straining (abdominal pressure). In apoplexies occurring during or after the act of delivery this is of essential importance. The cause of pregnancy-paralysis from cerebral hæmorrhage is not yet sufficiently clear; it occurs almost only in the second half or towards the end of the pregnancy (Hösslin [2]). Vomiting, *e.g.* in narcosis, has also been given as a cause of hæmorrhage. I have seen a few cases

[1] *R. n.*, 1901.

[2] *A. f. P.*, Bd. xxxviii.—see here also the literature.

in which, in severe vomiting of apparently gastric origin, cerebral symptoms (especially of a bulbar nature) immediately followed. Rad has published a similar case. But the explanation is by no means clear, for a cerebral origin of the vomiting cannot be definitely excluded. Again in narcosis-paralysis of cerebral origin softening processes are found, and on the other hand the action of the anæsthetic itself has been blamed (Büdinger, Schwarz, Cohn).

The above-mentioned factors of muscular strain and the forced expiratory movements have also to be considered as causes in those rare cases where cerebral hæmorrhage sets in during coitus—in a patient of Gumprecht's [1] taking a fatal course. Yet during this act embolic processes may also occur (Wagenmann, Oestreicher).

In the rare cases of hæmorrhage in childhood, such as have been observed in whooping-cough, the same factors seem to be in operation. It is true that the existence of hæmorrhages in the brain substance in this disease has been questioned, for, while meningeal hæmorrhages were found in some cases (Cazin, Kohts), in many others no pathological change could be discovered (Henoch, Luce, Kohts).

The exhaustive work of Neurath,[2] in which he collects and analyses all the published cases, shows that, in addition to inflammatory changes in the meninges, cerebral and meningeal hæmorrhages are important factors. Little is known regarding the hæmorrhages which arise from rupture of the veins. *Strong mental emotions*, e.g. fear and anger, are also exciting causes. It is doubtful whether these can cause rupture in a healthy cerebral vessel, but the possibility must be admitted. I have attended a strong young girl of sixteen years of age, who, on receiving the news of a death, fell down unconscious, and woke out of this condition with the signs of a cerebral hæmorrhage.

Hæmorrhages, the origin of which could not be clearly explained, have also been found in young persons, as in the recent cases of Pearce and Hunt.[3]

According to Volkmann the internal pressure must be increased fourteen times to cause a rupture of the normal carotid artery.

Cold baths and excess of alcohol are also to be mentioned as exciting causes of cerebral hæmorrhage. A strong young officer, previously healthy and not addicted to excess of alcohol, and in whom no signs of vascular disease could be detected, had an apoplectic seizure immediately after immoderate indulgence in drinking, the consequence of which was a permanent hemiplegia. The fact that cerebral hæmorrhage sometimes occurs during *sleep* is somewhat opposed to the above statements. Gowers is of opinion that the recumbent position may be the cause, the flow of blood from the brain being thereby rendered more difficult.

Grant thinks that strychnine (and quinine) should not be prescribed to persons with an apoplectic tendency.

Injuries to the skull mainly cause meningeal hæmorrhages but may also give rise to hæmorrhages into the brain substance. These are situated, as a rule, at the periphery of the brain, either in the cortex or sub-cortical white matter. According to Langerhans [4] the frontal lobe is most often

[1] *D. m. W.*, 1899.
[2] *Obersteiner*, xi. (Lit.).
[3] Pub. of Cornell Univ., 1904.
[4] "Die traumatische Spätapoplexie," Berlin, 1903.

affected. But the injury may also cause rupture of a miliary aneurism and thus give rise to hæmorrhage within the brain itself. In many cases there are multiple smaller foci, and the trauma moreover frequently causes a combination of meningeal with cerebral hæmorrhage.[1] The cranial contusion may be followed, even after an interval of days and weeks, by hæmorrhage, in the region of the aqueduct of Sylvius and fourth ventricle and also in the cortex, preceded, however, generally by a local tissue softening (*traumatic late apoplexy*). According to Duret and Bollinger,[2] this is due to the cerebro-spinal fluid being compressed on account of the cranial pressure, and producing at the places named destruction of the tissue. This is combined with traumatic degeneration and softening, and the vessels are also involved in the process to the extent even of a rupture.

Cases of this kind have been described by myself, by Matthes, Mazurkiewicz, Kolbe, Bruns, Bohne, Thiem, and others (*cf.* p. 749). Marie-Crouzon refer to "Apoplexie traumatique tardive," (*Revue de Méd.*, 1905). Some years previously Stadelmann discussed this subject very fully and advised that this condition should be diagnosed only after full consideration of traumatic late apoplexy. The diagnosis could be justified only if the individual had previously been healthy—free from arterio-sclerosis, nephritis, syphilis—and if the trauma was considerable. Langerhans has, however, taken up arms energetically against Bollinger's view and severely criticised the published observations: he affirms that they are not sufficiently supported by the post-mortem evidence; proof of the softening—to correspond with Bollinger's theory—is in particular lacking. Although we agree with the author in recognising that very great caution must be exercised in the diagnosis of a cerebral hæmorrhage appearing a long time after a cranial injury, so that a spontaneous hæmorrhage may not be confounded with a traumatic one, yet we think it right to maintain the conception of traumatic late apoplexy. Langerhans also admits that injuries to the skull may produce areas of red softening in the wall of the aqueduct of Sylvius.

Regarding the existence of so-called *vicarious* cerebral hæmorrhage there are few references in modern literature. In many older text-books it is stated that cerebral hæmorrhage may be brought about by the cessation or artificial suppression of the menses or by the stoppage of bleeding from *hæmorrhoids*, etc. I have seen one case which recalled this to my mind. An individual suffering from hæmorrhoids, who had lost much blood from this cause, was operated upon and became consequently free from hæmorrhoidal bleedings. A short time later hæmorrhages took place in the lungs and bladder, and as an apoplectic attack with paralysis. Although arterio-sclerosis was present, it was remarkable that, on the suppression of the hæmorrhoidal flow, hæmorrhages occurred in very varied parts. Another case, which I observed with Karewski, has remained quite obscure. About six days after an operation for hæmorrhoids in a young, strong man deep coma suddenly set in, from which he recovered after some hours; he has since remained healthy.

Suppurative processes in the skull, especially the petrous portion of the temporal bone, may lead to the development of cerebral hæmorrhage through the erosion into vessels and sinuses.

Regarding the spontaneous hæmorrhages due to angioma of the cerebral membranes see chapter on cerebral tumours and Cushing (*Journal of Am. Assoc.*, 1906).

Site of the Hœmorrhage.—Every part of the brain may become the seat of cerebral hæmorrhage, yet the separate regions of the brain are by no means affected to an equal extent. The *central ganglia* are the most common situations—the corpus striatum, the optic thalamus with the adjacent white strand of the inner and outer capsule, then the centrum semi-ovale, the cortex, and the pons. Cerebellar hæmorrhage is rare and hæmorrhage into the corpora quadrigemina and the medulla oblongata is even rarer. Here, however, it must be remembered that the medulla oblongata is a relatively small area.

[1] For details on this subject see Kocher, Nothnagel's "Handbuch," ix. 3, also Gebauer, *W. kl. R.*, 1903; Yoshikawa, *M. f. P.*, xx., Ergänz, and others.

[2] Festschr. R. Virchow, *Internat. Beitr.*, Berlin 1891.

In Wolfrum's paper (*Münch. med. Abhandl.*, i., 1904) the thalamus was affected in a hundred and three, the corpus striatum in fifty-seven, the internal capsule in fifty-four, and the lenticular nucleus in forty-eight cases.

Corresponding to the proportionate distribution of hæmorrhage in the various parts of the brain is the fact that miliary aneurisms are found most frequently in the arteries of the central ganglia and most rarely in those of the medulla oblongata. It is in the branches of the artery of the Sylvian fissure that enter into the cerebrum, viz., the lenticulo-striate and lenticulo-optic arteries, that aneurisms preferably develop (according to Durand-Fardel in 75 per cent. of the cases). These vessels, which arise at right angles to the arterial trunks, have a fairly large calibre, are *terminal arteries*, and remain under relatively high pressure. In contrast to these the vessels of the cerebral cortex after their commencement give off numerous branches, break up into a network, and possess at least minute anastomoses.

Morbid Anatomy.—Miliary aneurisms are just perceptible to the naked eye, their diameter varying from $\frac{1}{10}$ to 1 mm.; they appear as an ampulliform dilatation or bulging of the vessel, yet one may speak of aneurism only when the vessel-wall is diseased. The process arises in the muscular coat, which splits up and atrophies (Roth, Arndt, Loewenfeld [1]), and ultimately all the coats become degenerated and yield to the pressure.

The *size* of the hæmorrhage varies very much; it may reach the size of a man's fist and by rupturing into the ventricles may become still larger. The largest areas are found in the central ganglia and in the centrum semiovale, while in the cortex, cerebellum, and pons they are usually much smaller. The multiple foci found in general hæmorrhagic diatheses and in acute infectious diseases are not as a rule so large. Traumatic cerebral hæmorrhage is generally a circumscribed one (multiple punctiform); only exceptionally is it of some size (Langerhans). Within the hæmorrhage the blood is mixed with destroyed brain substance, and the adjacent cerebral tissue is torn up and traversed by small hæmorrhages.

According to the age of the focus it has the usual colour and characters of coagulated blood, or the blood effusion has already undergone transformation and assumed a dark-brown, later a yellowish tint. Up to about four weeks the blood focus usually contains a dense firm blood clot; in five weeks a yellowish discoloration is found (Monakow), and in two or three months it has an ochre-stained appearance. Microscopically we find fat globules, granular and crystalline *pigment* (hæmoglobin, hæmosiderin, hæmatoidin). From the discoloration, the swelling, the shrinking of the red blood corpuscles and their inclusion in contractile cells, and from the character of the pigment, one can estimate the age of the blood-effusion. On the third days cells, containing blood corpuscles, first appear. On the eighteenth day Dürck [2] first found pigment, and only on the sixtieth free pigment.

A longer time is necessary for the formation of *apoplectic* cysts: they are found, it is supposed, at the earliest not till three or four weeks. The adjacent cerebral substance forms, by proliferation of the glia and by connective tissue new formation, a kind of capsule around the focus, the contents of which are more and more absorbed until there remains only a cavity filled with serous fluid. It is seldom that any complete

[1] "Studien über Ätiol. und Pathol. der spont. Hirnblut," Wiesbaden, 1886, and *W. kl. W.*, 1887.
[2] *V. A.*, Bd. 130.

cicatrisation sets in (this is found more frequently after softenings).

If the hæmorrhage be extensive and death ensue at an early stage the convolutions of the affected hemisphere are found to be flattened, the sulci somewhat obliterated, and the falx cerebri pressed to the other side.

According to post-mortem statistics, hydrocephalus is a not infrequent phenomenon accompanying larger hæmorrhages (Wolfrum).

Symptoms.—Although the symptoms of cerebral hæmorrhage depend to a certain extent on the site and extent of the blood effusion, yet in almost every case two groups of symptoms may be distinguished—the first those of transitory, and the second those of permanent duration. The immediate result of cerebral hæmorrhage is *the apoplectic seizure*; the remote effect of cerebral lesion is paralysis, and as a rule unilateral paralysis—*hemiplegia.*

The Apoplectic Seizure.—The individual affected may suddenly and without any warning become unconscious and fall to the ground (apoplexie foudroyante). More frequently certain disturbances of the general health usher in the attack and warn the patient, at least a few minutes before the loss of consciousness. These premonitory symptoms consist in a feeling of giddiness, of determination of blood to the head, of intracranial pressure, of mental depression, of an alarming feeling of oppression at the heart; paræsthesia in one half of the body, mental confusion, and disturbance of speech may also occur. Retinal hæmorrhages and bleedings at the nose may also be prodromata. Such disturbances of the general health may go on for weeks before the attack, but this is unusual. The apoplectic seizure, as a rule, overtakes the individual while in good health.

The apoplectic, as he lies comatose, looks, on superficial observation, as if he were asleep, but he cannot be roused by any kind of stimulus. Voluntary movements and sensation are absent. The face is usually red and bloated; the pupils, which do not generally react to light, are of normal size or more usually dilated (contracted only in pontine hæmorrhages); the conjunctival—and corneal—reflexes are lost, the muscles are relaxed, the passively raised extremities fall as if lifeless, the deep reflexes tend to be absent during this stage, and the superficial reflexes are abolished. The pulse is for the most part strong and full, and either of normal rate, slowed, or, rarely, quickened. The respiration is usually slower and deeper than normal, frequently stertorous, as the inspiration sets in motion the relaxed soft palate and the loose vocal cords; more rarely the Cheyne-Stokes type of breathing is present. The cheeks are excessively puffed out during expiration. Fluid in the mouth is not swallowed. Urine and stools are evacuated involuntarily; the urine, the quantity of which may be increased, contains albumen and sometimes sugar (rarely for longer than 12 to 20 hours after the attack). General or partial convulsions seldom occur during an apoplectic attack.

The temperature falls 1° to 2° during the first hour, then rises to normal or somewhat above normal. A greater or more persistent fall of temperature, like a rapid or excessive rise of temperature, is of ominous signficance.

An attack of this nature may last from a few hours (1 to 4 on an average) up to one or two days.

The immediate cause of the coma is undoubtedly the disturbance of

the circulation (anæmia) in the cerebral cortex produced by the pressure of the extravasated blood and by other factors. The molecular vibration, also, which is transmitted from the seat of the hæmorrhage more or less through the whole brain, may cause an arrest of the cortical functions (this applies also to embolism). Monakow believes that contraction of the cortical arteries, produced reflexly, plays some part in the onset of the coma, and recently he has ascribed great significance to diaschisis. The deep coma usually gives place after a few hours to somnolence, and the corneal and other reflexes return; the deep reflexes can be elicited; the patient is able to swallow, makes slight voluntary movements, and thus reveals which side has been paralysed.

This phase of the *attack*, as a rule after 24 to 48 hours, passes over into one of *reaction* (corresponding to the reactive processes in the blood focus and its neighbourhood); the pulse becomes more frequent, the temperature rises to normal or above it, the skin is usually bathed in perspiration; headache, slight delirium, sensory and motor signs of irritation may set in. These disturbances are generally of brief duration.

The apoplectic attack does not by any means always go hand in hand with a condition of deep coma. Sometimes there is simply loss, or mere dulling of consciousness, while the signs of coma are absent. A transitory giddiness or a quickly passing feeling of stupor may even take the place of the apoplectic seizure, and finally, the paralytic phenomena may make their appearance without the slightest impairment of consciousness. These variations are closely dependent upon the extent of the hæmorrhage, the rapidity with which it occurs, and the path of its propagation. When, as is usually the case, the extravasation of blood takes place rapidly and is larger than a cherry or a hazel-nut, it is marked by a more or less considerable disturbance of consciousness.

Cerebellar or pontine hæmorrhages more frequently occur without loss of consciousness than those which affect the cerebrum.

Among the group of *irregular* symptoms which accompany apoplectic attacks are *vomiting* (which is most frequently observed in cerebellar hæmorrhages), and deviation of the head and of the eyes to one side—in cerebral hæmorrhages towards the side of the focus. General and unilateral *convulsions* and delirium only exceptionally inaugurate or accompany an apoplectic seizure.

Changes in the fundus oculi—papillitis or choked disc—practically never occur in simple cerebral hæmorrhage, but they are always present in meningeal hæmorrhage resulting from the rupture of a basal aneurism, and also, though very seldom, in large cerebral hæmorrhages which penetrate towards the base (*cf.* pp. 713 and 747). The affection of the eye is, therefore, largely a unilateral one and corresponds to the side of the hæmorrhage (Mackenzie, Michel, Uhthoff, and Oppenheim). An observation of Nonne's shows that in exceptional cases choked disc may develop in cerebral hæmorrhage. Here perhaps the active rôle is played by the combination with hydrocephalus suggested by Wolfrum.

When the patient does not die during the attack but gradually recovers consciousness it is found that the apoplectic focal symptoms are left behind. Of these the most frequent is a paralysed condition, viz., hemiplegia. Even before the patient makes any movement, the side of the body affected can usually be recognised. By careful observation an asymmetry can be seen in the face; the angle of the mouth is depressed on the paralysed side

and the cheek puffed out, the naso-labial fold is less marked or quite effaced, and the saliva flows out on this side. If the extremities are passively raised, in complete coma they fall on both sides as if lifeless, but as soon as the stupor is lessened it can be ascertained that the tonicity of the muscles again confers a certain tone upon the extremities of one-half of the body, while those of the other are quite flaccid. One must, however, beware of confusing signs of motor irritation with voluntary movements. The condition of the reflexes also gives a clue—especially the *cremasteric* and *abdominal* reflexes, which are absent on the paralysed side (Rosenbach, Jastrowitz). So also, even during the apoplectic seizure or immediately after it, the Babinski and Oppenheim signs can frequently be found on the paralysed side (Babinski, Brissaud, Oppenheim). The Bechterew-Mendel reflex would also be of some assistance.

The condition of the respiratory muscles may itself in many cases indicate, even during apoplectic coma, the site of the paralysis, as the corresponding half of the chest remains quiet during respiration (Nothnagel, Grawitz, Ortner), and the diaphragm moves much less forcibly. Yet, according to the observations of Jackson and Weisenburg (see p. 685), this applies only to deep breathing, while in ordinary respiration the condition is reversed.

If conjugate deviation be present, then the seat of hæmorrhage is almost always the side towards which head and eyes are turned.

Gradually voluntary movements return in the limbs of one-half of the body. Manipulations with either the arm or leg, *e.g.* placing it in an uncomfortable position, show that it can voluntarily be brought back from this position, in contrast to the absence of such power on the paralysed side.

If there is no apoplectic seizure, then the paralysis is the first sign of disease; its onset is perceived by the patient himself, for the object which he holds falls from his hand, his leg bends, and he must sit down or it gives way.

The typical result of this attack is *hemiplegia*, *i.e.* paralysis of the arm, leg, and parts supplied by the facial and hypoglossal nerves on one side of the body (see p. 656).

The muscles of the pharynx, larynx, and trunk almost always escape —the trapezius alone tends to share in the paralysis.

At first after the attack the sensibility is usually blunted on the paralysed side of the body. The speech is somewhat indistinct: slight disturbance in articulation is present, which for the most part has already almost or entirely disappeared in a few days.

This constitutes the clinical picture in the great majority of cases, where the hæmorrhage is found in the usual position,—the region of the central ganglia,—and has injured, either directly or by pressure, the motor tract in its course through the posterior limb of the internal capsule. If this is *directly* involved by the blood-extravasation, and is more or less completely destroyed, then the hemiplegia persists—forming a *direct* focal symptom.

Later, however, the clinical picture undergoes certain changes: soon—sometimes a few days or even a few hours after the attack—an increase of the deep reflexes on the paralysed side is observed and ankle-clonus may be obtained. This increase of the deep reflexes is frequently present—although to a lesser extent—in the extremities, especially the leg,

of the sound side; as a matter of fact a slight diminution of the motor power also in the muscles of the extremities of the sound side is usually found. In a few cases I have noted, as the expression of this bilateral increase in the deep reflexes, the so-called jaw-jerk.

Ganault (*cf.* p. 688) has investigated the condition of the deep reflexes in hemiplegia. He found in hemiplegia of long duration the knee-jerks increased on the paralysed side in 92 per cent. of the cases, in 6 per cent. both sides alike, and in 2 per cent. weaker on the paralysed side than on the sound side. On this side it was normal in 60 per cent. and increased in 25 per cent. of the cases.

In the paralysed arm the deep reflexes were almost always increased, and, through percussion of the bone, contractions could be brought about in parts where usually no muscular twitching could be elicited, *e.g.* from the metacarpus, styloid process of the ulna, etc. It is unnecessary to regard each of these contractions as Bechterew does, as a reflex requiring a special name. Parhon-Goldstein, Böttiger (*N.C.*, 1902), and others have recently made communications on this subject.

After a few weeks a certain degree of movement returns in the paralysed leg, and gradually the patient regains command over it so that he can raise it from the horizontal to an elevated position, and especially can extend the leg—though only slowly and with diminished strength. The arm (in severe cases) remains either quite paralysed or there are slight movements which the patient again learns to make — usually slight abduction of the upper arm, slight flexion of the elbow, and a few slow, ineffectual finger-movements. The paralysis in the extension of the hand and fingers persists to a great extent, whilst in flexion a certain degree of movement returns, so that the patient can close the hand but not open it. In the leg, flexion of the knee and extension of the foot and toes are affected for a longer time (Wernicke, Mann). In any case the arm is more severely affected than the leg.

This is the usual condition, but there are exceptions. Thus hæmorrhages which have their site in the cortex of the motor zone, or in the corresponding meningeal area or in the sub-cortical white matter related to it, may affect the leg more severely than the arm. The leg may be also specially affected when, in addition to the cerebral hæmorrhage, a lesion in the spinal cord involving the pyramidal tract is present. I have noted this with special frequency in syphilis. In hæmorrhages into the pons and medulla oblongata I have occasionally found that the paralysis of the leg or legs is greater than that of the arm. This also applies to diplegia.

The hemiplegic almost invariably recovers the power of walking, although only after a few months—in the cases of average severity six to eight weeks (Gilles de la Tourette)—whilst the powerlessness of the arms generally continues.

But other disorders—muscle retraction and shortening—*contracture*—almost invariably occur. The arm in particular is subject to this, and a definite fixed position results in almost every case: the upper arm is adducted, the lower arm bent at a right or an acute angle, the hand usually pronated and also slightly flexed, the proximal phalanges slightly flexed, the middle and terminal phalanges more so. If an endeavour is made to alter the position of the arm, the muscle tension has to be overcome: this may be wholly or only to a certain extent successful, but the arm immediately returns to the former position. If the finger is moved from the flexed to the extended position, this succeeds, as a rule, only after the hand is placed in extreme flexion.

The deformity occasioned by the contracture is less evident in the

leg because this is fixed in extension. Exceptions are rare—flexure of the leg arises now and then when the patient is long confined to bed.

The contracture of the Achilles tendon, and the pes equinus and *equino-varus* positions of the foot resulting from this, gives to the extremity a characteristic aspect and forms a very real hindrance to walking. The patient rests more firmly upon the sound leg, draws the paralysed one after him, and circumducts it—letting the foot describe a semicircle while he raises the pelvis on the paralysed side and carries it from behind forwards in a circle, which is described with the passive leg as an axis. The toes are dragged along the floor.

When the patient raises himself from the recumbent position the paralysed leg is strongly flexed at the hip-joint, and is lifted from the horizontal position at the same moment as the trunk is raised (Oppenheim,[1] Babinski [2]). This sign is by no means always distinctly marked, and it appears on the other hand even in health. When the trunk is raised from the horizontal position the legs are elevated. In health, of course, this can be overcome.

The paralysed arm is, during walking, usually supported by the sound one, especially if there is any tendency to associated movement. Contracture very rarely appears in the muscles supplied by the facial nerve. In longstanding hemiplegia one sometimes notes that the mouth is drawn towards the paralysed side and the naso-labial fold is deeper on this side.

In rare cases in the stage of contracture the tongue is affected in such a way that when protruded it deviates to the side opposite to the hemiplegia (Minor); I have only very rarely seen this.

It is the rule that contracture is associated with persistent hemiplegia, but there are exceptions. In very rare cases the resultant paralysis is flaccid without there being any explanation for this divergence from the usual condition. I have found this condition especially in very extensive foci which involved both the motor and sensory areas. It has already been noted that signs of motor irritation of other kinds may in individual cases appear in the paralysed limbs (see p. 690).

The muscles usually retain their normal size or undergo, in consequence of inactivity, a moderate atrophy with slight quantitative reduction of the electrical excitability (see p. 689). The statements to the contrary of Marinesco, Parhon-Goldstein, De Grazia, etc., are not in a line with my experience.

If the hæmorrhage has not directly affected the motor tract but has injured it only by pressure, the hemiplegia is not permanent. It is recovered from either very rapidly or within a few weeks or months, and is therefore merely an *indirect focal symptom*. It is conceivable that between these two varieties—the direct and indirect involvement of a conducting tract—there is no sharp boundary: the extravasated blood might, *e.g.*, be situated in the optic thalamus or in the lenticular nucleus, and certainly act by pressure, and yet a portion of the motor fibres may be destroyed.

Lately Rothmann and Lazarus have suggested the view that, after the destruction of the pyramidal tracts, other tracts of fibres—the so-called extra-pyramidal tracts—become more active and can, up to a certain extent, take over the function of the first. According to our experience no actual compensation takes place in this way.

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[1] *Charité-Annalen*, xiv., 1889, and *M. f. P.*, xi.

[2] *Gaz. des hôp.*, 1900.

If the hæmorrhage extends to the most posterior part of the internal capsule or into the optic thalamus (rupture of the lenticulo-optic or choroidal arteries), then *hemianæsthesia* is associated with the hemiplegia. This is rarely complete, and is usually more or less a kind of dulled sensibility—which may extend to all kinds of stimuli or only to individual ones. It is found over the whole affected side or only on certain parts (especially the distal portions of the extremities).

In rare cases the hæmorrhage is limited chiefly to the region of the sensory tract. There is then complete hemianæthesia, while the hemiplegia is absent or rapidly recovers or is distinctly marked only in the leg. Then another derangement of movement appears, viz., hemiataxia, which probably is a result of the anæsthesia, especially of loss of deep sensibility.

Signs of sensory irritation in the paralysed or anæsthetic limbs are rarely complained of. *Pain* may set in in consequence of joint-changes, which arise through inactivity and are only exceptionally the result of trophic disturbances. This is most frequently present in the shoulder-joint, and is in part to be traced to muscular over-strain, since the arm hanging as a dead weight on the trunk affects the shoulder muscles. But the pain may also be a direct consequence of cerebral disease, as is pointed out on p. 707.

Regarding the condition of the bladder-functions, see p. 651.

A certain degree of alteration in the mental functions is always caused by hæmorrhage that is at all extensive: simple diminution in intelligence, irascibility, irritability, failure of memory, etc. It is obviously difficult to say how much of this is to be ascribed to the extravasation itself and its consequences, and how much to an existing affection of the cerebral vessels. Greater degrees of dementia—a true dementia apoplectica—develop apparently almost only in encephalomalacia, owing to a general atheroma, and especially after apoplectic seizures of syphilitic origin. Traumatic hemiplegia is frequently combined with psychic derangements—especially with changes in character (René Martial [1]).

Vaso-motor disturbances are not unusual. In the beginning the temperature of the paralysed limb is often raised, while in the later stages the skin on the paralysed extremities feels cold and is cyanotic.

Increase in the blood-pressure on the paralysed side was found in recent hemiplegia by Parhon-Papinian (*Spitalul.*, 1904). Respecting œdema in hemiplegia, see p. 710.

Trophic disturbances must be looked upon as the cause of certain joint-changes, which develop—though rarely—some weeks after the attack and are associated with swelling, redness, and pain in the joint. Hæmorrhages into the joints are of rare occurrence (Obici). Those joint changes which are occasioned by contracture and immobilisation have already been referred to. *Acute decubitus* should also be included here; *i.e.* a decubitus spreading very rapidly a few days or a week after the onset of the hemiplegia, and affecting the gluteal region of the paralysed side. According to Charcot's description redness first appears, which is followed by a petechial, dark-violet discoloration; then vesicles form which become confluent, and ulceration and the development of an extensive, dry, gangrenous eschar next appear. Some authors (Brown-Séquard, Charcot, Ollivier, Hunnius) relate the pneumonia,

[1] *Thèse de Paris* and *Nouv. Icon.*, xiii.

which sometimes appears in apoplectic patients, to the cerebral affection, and regard it as the expression of vaso-motor trophic disturbances. Hæmorrhage into the stomach has been similarly explained (Charcot, Ebstein [1]).

In describing the symptoms we started with those of the typical localisation. It is natural that the symptoms of the attack undergo an essential modification when the hæmorrhage is in another part of the brain.

If the *centrum semiovale* be affected, then, in hæmorrhages of great extent, hemiplegia may be a direct or indirect focal symptom. Smaller ones may occur without any symptoms resulting. If the hæmorrhage is in the *central ganglia* of the *left* hemisphere or in the left internal capsule a circumscribed area might cause a temporary aphasia : the more extensive the hæmorrhage and the nearer it approaches the speech centre, the more marked and more persistent will be the aphasia. The variety of symptoms caused by the variable localisation of the hæmorrhages need not further be discussed. If the hæmorrhage is in the cortex —a very rare occurrence—and if the motor region is affected, unilateral tonic and clonic spasms may be present.

Ventricular and pontine hæmorrhages require special consideration. *Primary ventricular* hæmorrhages are very rare. Far more frequently the extravasated blood, situated in the adjacent white matter of the brain, ruptures into the ventricles (usually first into the lateral ventricle), and gradually invades all the cavities of the brain. This process causes very striking symptoms : the insensibility is deepened ; if there had been no loss of consciousness, or if it had been regained, then a new apoplectic seizure is added—the paralysis extending to all four extremities ; convulsions (general or unilateral on the side which to begin with was unaffected) set in, or more frequently, rigidity in the muscles of the hemiplegic side or in all four extremities. To these are usually added slowing of the pulse, fall of temperature, and considerable respiratory disturbances. The diagnosis may be assisted by lumbar puncture—showing a large amount of blood in the cerebro-spinal fluid, as is proved by the observations of Dupré-Sébilleau, Ohm,[2] and others. Before death, which almost inevitably occurs—indeed within the first twenty-four hours [3]—the pulse becomes small and rapid, the pupils, which were at first contracted, become dilated and fixed, and cyanosis and asphyxia develop.

Hæmorrhages into the pons and medulla oblongata may extend irregularly, involving both sides, or be limited to one side if arrested by the raphé (Gowers). Sometimes they are situated in the medial region—the raphé and its neighbourhood—and these, specially, tend to cause the most severe and most extensive lesions (Mickle,[4] Willcocks). Others spread rather in the tegmental area as far as the floor of the fourth ventricle, and others tend rather to pass downwards to the lower levels of the pons. They may have a considerable horizontal extension. Extravasation of blood into the pons is not always associated with loss of consciousness ; the apoplectic attack may here be replaced by a simple attack of vertigo. From the published discussions of this subject (in

[1] In regard to this subject see Gaumé, "Contribution à l'étude des Hémorragies viscérales second. aux Hémorragies encéphal," *Thèse de Paris*, 1903.

[2] *D. m. W.*, 1907.

[3] In Jones' experience (*Br.*, 1905) death occurs within the first twenty-four hours in 65 per cent., and within a week in 90 per cent. of the cases.

[4] *Brit. Med. Journ.*, 1881.

addition to those already mentioned, the observations of Gull, Schütz, Meyer, Senator,[1] Rochefontaine, Joffroy, Raymond, Dutil,[2] Russell, Gee, Tooth,[3] Bruce, Luce,[4] Gumprecht, Elsholz, Clerc, Queirolo, Wallenberg, Cohn,[5] Dana,[6] etc., must be specially named), we may deduce the following facts as regards the symptomatology: the hæmorrhage has frequently been so rapidly fatal that localising symptoms could not be ascertained. *Hemiplegia alternans*, in which the cranial nerves are often bilaterally affected, is a frequent symptom; and not infrequently paralysis of both sides of the body is either present from the outset or develops during the course of the illness. The paralysis is usually associated with disturbances of articulation and deglutition. The pupils are usually contracted and do not react to light—a symptom which even in coma gives a valuable indication: but they may also be dilated and rigid. The external muscles of the eye and the muscles of mastication, etc., are often affected by the paralysis, and the sensory trigeminal nerve may often participate. Unilateral or general convulsions are very frequent, and trismus or more rarely opisthotonos may occur (Lépine). The temperature tends to rise rapidly and considerably—as high as 40° C. (104° F.) and upwards—and the respiration is altered (Cheyne-Stokes breathing, simple slowing or acceleration, and irregularity). Luce, on the strength of his own observations and the consideration of the views of others, inclines towards Nothnagel's teaching that the pons contains a centre for convulsions: he even thinks that epilepsy may have its origin in the motor nuclei of the pons. He has found that the muscles of the trunk and eyes especially share in the convulsions which have their origin in the pons.

Hæmorrhages into the pons may, however, produce much less marked symptoms (*cf.* chapter on acute bulbar paralysis). In a case observed by Elsholz, for instance, the paralysis was limited to a number of cranial nerves.

In hæmorrhage into the medulla oblongata, if death does not immediately ensue, grave disturbances of circulation and respiration develop along with signs of bulbar paralysis.

Cerebellar hæmorrhages, which are very rare—Touche,[7] Boldt,[8] Starr,[9] Witte,[10] recently described cases of this nature—produce corresponding symptoms of cerebellar ataxia, vertigo, vomiting, etc. Opisthotonos may also develop (Thyne [11]). A similar group of symptoms results from hæmorrhage into the superior cerebellar peduncle, as has been shown by the excellent observations of Porot.[12]

The corpora quadrigemina are very rarely the seat of primary cerebral hæmorrhage—see case of Bouchaud.[13] Paralysis of the eye muscles, disturbances of equilibrium, and diminution in the acuity of hearing, etc., may be the chief localising symptoms.

With regard to "hemiplegia without pathological changes," see next chapter.

Differential Diagnosis.—It is of extreme importance to make a correct diagnosis during an apoplectic attack. Many difficulties, some of them very great, have to be overcome in distinguishing it from conditions of simple loss of consciousness, epileptic stupor, hysteria, uræmia, coma, and other toxic forms of disturbance of consciousness.

[1] *A. f. P.*, xiv.
[2] *Gaz. méd de Paris*, 1887.
[3] *Br.*, 1898.
[4] *Z. f. N.*, xv. (Lit.).
[5] *A. f. P.*, xxxiv.
[6] *Med. Record*, 1903 (Lit.).
[7] *Arch. gén. de Méd.*, 1900.
[8] *D. m. W.*, 1905.
[9] *Med. Record*, 1906.
[10] *N. C.*, 1906.
[11] *Lancet*, 1901.
[12] *R. n.*, 1906.
[13] *Arch. gén. de Méd.*, 1903.

Simple syncope is usually easily diagnosed; here the loss of consciousness is rarely complete—the signs of coma are absent—and the attack lasts as a rule only a short time. The chief distinguishing feature is that the action of the heart is involved, and the pulse is small and often accelerated. The individual appears pale and stupefied, but not comatose.

Regarding the "Lachschag" (laughing attack) which I have described, see p. 673. The *epileptic* attack in itself has no resemblance to the apoplectic. The somnolent condition which follows or replaces it, may, however, when there is no history in the case, lead to hesitation in the diagnosis. If convulsions have preceded the attack, then the mark of a bite of the tongue or a scar resulting from former attacks point to the epileptic nature of the condition. If the coma is an expression of an epileptic attack the pallor of the face is striking. Unilateral symptoms are seldom present in attacks of true epilepsy, and, further, epilepsy appears, as a rule, at an early age.

The congestive attacks of general paralysis may entirely resemble the apoplectic: the distinction can be made only by referring to the history of the case, or by watching its further progress. This applies also to the apoplectiform attack of multiple sclerosis. When young individuals with healthy heart and blood-vessels are seized by a rapidly passing attack, the possibility that it may be of a symptomatic nature should always be borne in mind.

Hysterical conditions of apparent or actual loss of consciousness seldom give rise to mistake. The pupil-reflex, and usually the corneal reflex also, are conserved, and the deep reflexes are also retained. The toe and leg reflex are normal. The pulse may be accelerated, but is never actually slowed. The expression of the face shows that the patient is under the power of an illusion or of a morbidly intensified emotion. The temperature is neither lowered nor, except in very rare cases, elevated. Finally, it is often possible by means of ovarian pressure or psychic influence to relieve or modify the attack.

Uræmic coma cannot without further confirmation be recognised by investigation of the urine alone—as albuminuria may accompany the apoplectic attack. A microscopic examination may often lead to a definite diagnosis. In coming to a decision it is important to note the existence of œdema or albuminuric retinitis, and to ascertain if there is any previous history of other symptoms of uræmia (convulsions, amaurosis, vomiting, asthmatic conditions, etc.). The temperature is almost invariably subnormal. Uræmic coma hardly ever develops suddenly, but is ushered in by vomiting and convulsions. If contracted kidney exists, the conditions are equally favourable to the production of hæmorrhage and uræmia, and the diagnosis must therefore be reserved during the coma. *Diabetic coma* can usually be recognised without difficulty from an investigation of the urine, consideration of the general condition, and the smell of acetone. An attack of *encephalopathia saturnina* is generally associated with delirium and convulsions: the history of the case, and other signs of chronic lead-poisoning, very rarely leave any doubt as to the diagnosis.

In recent literature hardly any reference is made to the so-called *apoplexia serosa* on which in former times much stress used to be laid. It is, however, worthy of observation that here and there in individuals afflicted with paralysis, *i.e.* with the symptoms of an apoplectic attack, cerebral œdema was found, instead of the expected hæmorrhage, *e.g.* in a case of Bouveret's (*Revue de Méd.*, 1899), in which, along with severe hæmorrhages from the stomach, aphasia and

hemiplegia developed, with signs of apoplexy, and the patient died on the tenth day. Barr (*Brit. Med. Journ.*, 1902) understands by serous apoplexy a meningitis serosa ventricularis of acute and apoplectiform onset.

Grave alcoholic intoxication may be recognised by the spirituous odour, the nature of the vomit, the inclination to delirium, and the general restlessness. But it must be borne in mind that cerebral hæmorrhage is not infrequently associated with alcoholic excess. In acute *morphia-poisoning* the pupils are contracted *ad maximum*.

Acute hæmorrhagic encephalitis is frequently ushered in with unconsciousness, but marked premonitory symptoms are present (headache, psychic anomalies, etc.). The temperature is mostly raised, the coma is not so deep as in the apoplectic attack, and, in particular, the superficial and deep reflexes are retained.

The practical importance of deciding whether *cerebral hæmorrhage* or *cerebral softening* is present, cannot be overrated. It is by no means always an easy decision.[1] The employment of lumbar puncture for this object, as has been proposed and tried by Kroenig,[2] is to be strongly deprecated. Cerebral hæmorrhage is rare before the fourth decade, while embolism is frequent in youth, and thrombosis occurs almost solely in later years. Any cardiac defect makes embolic softening very probable: in atheroma of the heart, thrombosis is the most likely condition, although an embolus may arise from an atheromatous heart. Further, simultaneous disease of the cerebral vessels may cause cerebral hæmorrhage. When there is a simple hypertrophy of the left ventricle, especially if combined with nephritis, the case is, as a rule, one of cerebral hæmorrhage. If acute articular rheumatism has been previously present then, even in the absence of any lesion of the heart, we must still think of an embolism, as the endocarditis may have been cured. If the face is red and bloated, the pulse full and powerful, slow and of high tension, a diagnosis of cerebral hæmorrhage is justified. It must, however, be kept in mind that even in cerebral hæmorrhage the face may be pale and the pulse soft and weak. The condition of the blood-pressure may give valuable assistance in the differential diagnosis (Arullani). The coma in cerebral hæmorrhage is usually deeper and of longer duration than in softening. If very marked atheroma of the peripheral arteries is present, then cerebral softening is the more probable condition. Syphilitic endarteritis more frequently leads to thrombosis than to rupture of the vessels. Premonitory symptoms—paræsthesia, transitory feelings of weakness in the side subsequently paralysed, intermittent dysarthria (Huyghe[3])—which precede the attack for weeks, argue in favour of a softening due to thrombosis, while in embolism almost all prodromata (as regards the brain) are absent, though they are frequently also lacking in cerebral hæmorrhage. Conditions of mental weakness often for a long time precede the commencement of thrombosis, just as on the other hand they frequently accompany or succeed it.

Recurrence of the apoplectic attack points specially to softening. For the diagnosis of embolism it is of special value if infarcts have already formed in other organs (lungs, kidneys, etc.), or if embolism of the central artery of the retina has occurred simultaneously with

[1] Some time ago Friedenreich, *Hosp. Tid.*, 1906, discussed this subject again with the aid of more material.

[2] *L.c.* See further Crouzon, *R. n.*, 1903; Dupré-Sébilleau, *R. n.*, 1903.

[3] *R. n.*, 1905.

the cerebral hæmorrhage. Even in atheroma, thrombosis of this vessel may develop.

Any considerable fall or rise of temperature during the attack is evidence in favour of hæmorrhage. In ulcerative endocarditis, it is true, embolism may occur with fever and rigors.

In the next chapter we shall discuss the subject of the so-called lacunar porosis (Marie, Ferrand) and its relation to senile hemiplegia.

If the symptoms of paralysis are already present, or if the attack has passed or has been completely absent, the above-mentioned factors must be taken into account in making the diagnosis. The following points must, however, be specially considered : if there has been no loss of consciousness and the resulting symptoms on the other hand point to an extensive lesion, it is in the highest degree probable that they are caused by the softening process. This assumption is also justified when only a short lapse of consciousness or an attack of vertigo has occurred. An improvement beginning in the first days of the attack and steadily advancing makes the hæmorrhagic nature of the process probable.

The symptomatology of hæmorrhage differs, therefore, considerably from that of *cerebral tumour*, yet there are occasional cases in which the cysts resulting from the hæmorrhage produce focal symptoms which correspond to those caused by a neoplasm. In these cases we have usually to do with traumatic hæmorrhages in the cortex (or meninges) in the motor area, the origin of which could often be traced back even to childhood. Yet the symptomatology differs decidedly from that of tumours in the absence of the significant symptom of cerebral pressure. On the other hand a cerebral tumour may remain latent until a hæmorrhage within it gives rise to symptoms of an apoplectic seizure with hemiplegia. The presence of choked disc therefore justifies the diagnosis of tumour or at least makes its existence probable.

Even the clinical picture in apoplexia ingravescens has only an apparent similarity to that of cerebral tumour, as in a case described by Touche (*R. n.*, 1902) in which the large blood-extravasation in the parietal and temporal lobes had led to hydrocephalus and through this caused death.

As regards the so-called progressive hemiplegia, see next chapter.

Hysterical hemiplegia is, as a rule, easily distinguished from that due to cerebral hæmorrhage or any organic cause. It usually spares the areas supplied by the facial and hypoglossal nerves, or these participate in a quite unusual manner (see chapter on hysteria). Hysterical hemiplegia may be flaccid or associated with contracture. In the latter case the contracture has all the characters of hysteria (*q. v.*). The deep reflexes may be increased to the stage of clonus, but the exaggeration frequently disappears if the attention is diverted and its psychical origin is thus revealed : usually too it can be ascertained that the ankle clonus is not a consequence of the stretching of the Achilles tendon. Thus I have sometimes found that it was elicited as markedly in plantar as in dorsal flexion. The Babinski and Oppenheim signs, which are frequently present in organic hemiplegia, are almost invariably absent in hysteria. The Bechterew-Mendel sign is likewise to be ascribed to true hemiplegia. On lifting the arm in the supine position the hand of the paralysed side falls into the position of pronation (Strümpell)—a symptom which also

occurs only in true hemiplegia. The tibialis sign (Oppenheim) may also be used for diagnosis, though this sign has only a limited value.

The cremasteric and abdominal reflexes are usually absent on the paralysed side in true hemiplegia, but are present in hysteria. The gait in true hemiplegia is modified in a characteristic manner, as has been described above, but in hysteria the leg is simply dragged along without circumduction: it hangs for a time in the air or touches the ground with the whole plantar surface.

Schüller has pointed out with regard to this that in walking sideways the genuine hemiplegic goes more easily towards the affected than towards the sound side. This is confirmed by Campbell and Crouzon, but I have not been able to convince myself that this sign is of any real importance.

Babinski, who has written a valuable treatise (*Gaz. des hôp.*, 1900) upon the differential diagnosis of organic and hysterical hemiplegia, rightly lays stress upon the fact that the platysma is occasionally involved in true hemiplegia. This is the result of the participation of the facial nerve in the paralysis.

Finally, hysterical hemiplegia is almost always accompanied by a characteristic hemianæsthesia and the other stigmata of hysteria. A physician, suspecting the nature of such a case, can generally by some method of suggestion ascertain the psychic basis of the paralysis.

I have observed a few cases in which rapidly passing hemiplegia and other focal brain symptoms were present, and could only be due to *vaso-motor* disturbances in one of the cerebral arteries, although there was no hemicrania. Communications regarding transitory hemiplegia of this character have been made by Russell,[1] Langwill,[2] Edgeworth, and others. Obviously in such cases the vessels were diseased, but there must in addition have been vaso-motor excitement in order to cut off the blood-supply temporarily. We must remember in this connection the processes within the sphere of the cerebral arteries which cause intermittent claudication.

Prognosis. 1. During the attack. The apoplectic seizure may lead to immediate death. Danger to life depends upon the site of the hæmorrhage and rupture into the ventricle. The duration of the coma gives a valuable indication as to the size of the hæmorrhagic focus. If it last for more than twenty-four hours, then life is endangered, although in exceptional cases recovery may take place after two days. A considerable and persistent fall of temperature is serious, likewise an excessive rise up to 40° C. (104° F.) and over. Lépine's case proves that there are exceptions to this rule. The gradual appearance of a constantly deepening somnolence (apoplexia ingravescens) is to be regarded as a "signum mali ominis," and this is to a greater extent true of acute bed-sore. Conjugate deviation points to a large focus, but is not in itself an indication of danger to life. Cheyne-Stokes breathing is a serious sign. Nephritis makes the prognosis more grave. Williamson states that, in cerebral hæmorrhage with fatal termination, retinal hæmorrhages are specially frequent on the side corresponding to the hemisphere affected.

If symptoms set in which point to a rupture into the ventricles, then the prognosis is most grave. Hæmorrhage into the pons usually runs a fatal course, and that into the medulla almost invariably so; the appearance, therefore, of bilateral paralysis is always serious. Death may occur, as in primary ventricular hæmorrhages, in a few minutes. *Pneumonia* is a further danger.

[1] *Practitioner*, 1906. [2] *Scot. Med. Journ.*, 1906.

2. During the stage of paralysis. If hemiplegia or any other group of symptoms has developed as a result of the hæmorrhage, it is necessary to ascertain whether these symptoms will retrograde, and whether improvement or complete recovery is to be expected. The depth and duration of the apoplectic seizure are important factors in the prognosis, as they indicate the extent of the hæmorrhagic focus. The more incomplete the apoplexy, the better in general are the chances of recovery. A small hæmorrhage may, however, be sufficient to completely interrupt the motor tract when it has its site in this tract. It must be ascertained, therefore, whether the hemiplegia and the related manifestations are *direct* or *indirect* focal symptoms. The course during the first few weeks will decide this point. If, during the first month, the hemiplegia continue unchanged in intensity, or if only a trace of movement sets in, in the affected leg, it is almost certainly a direct focal symptom and will show only the improvement mentioned above. On the other hand a certain degree of movement setting in during the first few days, and steadily advancing during the first period, is a very favourable sign. Exaggeration of the deep reflexes does not of itself affect the prognosis, nor does the Babinski sign. As soon, however, as the first evidence of contracture sets in, there is hardly any prospect of complete restoration.

Aphasia, when associated with hemiplegia, and a correct diagnosis of cerebral hæmorrhage, gives, on the whole, a relatively favourable prognosis. Speech is usually restored, although for complete recovery a long period is requisite. A partial aphasia may persist. Should hemianopsia not improve within the first few days, it is likely to be a permanent symptom. Yet I have seen more or less complete recovery from the hemianopsia in traumatic cerebral affections associated with hæmorrhage.

The rapid disappearance, within a few days, of the symptoms of paralysis left by the apoplexy suggests paralytic dementia; careful investigation will almost always give a decisive answer on this point. It is only when disorders of the intelligence are not very marked, when the pupil reflex is retained, and the characteristic disturbance of speech is absent, that it may be difficult and even impossible to make a definite diagnosis. Dementia, following the attack and lasting for a considerable time, is an unfavourable sign.

If the hemiplegia is a direct focal symptom, the patient is able, as a rule, to walk again, but it is at least three to four months before he recovers this power. If the hemiplegia is a consequence of pressure it may completely disappear within a few weeks: yet here, too, a period of a few months is necessary for complete restoration.

Apoplexy coming on during parturition has an unfavourable prognosis (Hösslin). For the prognosis of uræmic and diabetic hemiplegia, see next chapter.

The life of those who have recovered from cerebral hæmorrhage is in future endangered from the fact that the conditions necessary for a repetition of the attack are largely present. But, on the other hand, I have known—apart from children with cerebral paralysis—individuals who have lived twenty to thirty years with their hemiplegia, or after their recovery, without another attack. The fact stated by Ballet and Dutil that old hæmorrhagic foci form areas into which infective agents make their way and settle, seems to have no real practical interest.

Treatment.—Prophylatic measures must first be taken to prevent the

development of disease of the vessels, and, if this be present, to remove it or render it harmless. Our power in this respect, however, is very limited. The most important point is the avoidance of alcohol and the other poisons previously mentioned. There are no means known to us of checking the formation of miliary aneurisms. As corpulence seems to favour the onset of cerebral hæmorrhage, suitable diet must be prescribed. For the arterio-sclerosis, preparations of iodine are usually prescribed: some physicians, *e.g.* Schrötter, have opposed their use, but the experimental results of Korànyi (*D. m. W.*, 1907) and Boveri indicate their value. Lauder Brunton prescribes the regular use of alkalies (Pot. bicarb. 1·8 gram.; pot. nitrate 1·2 gram., and sod. nitrite 0·003 gram., to one-half litre of water), and under the name "antisclerosin," a preparation of this kind is now sold. Rumpf recommends a diet deficient in lime, particularly avoiding milk, cheese, and eggs, etc.; he also prescribes lactic acid. Huchard on the other hand recommends an abundant supply of milk—this certainly is more in keeping with general experience and with my own—while he prohibits fish, game, etc. Romberg [1] prefers to the régime recommended by Rumpf and Huchard, that the patient himself should regulate his diet by his own experience, avoiding excessive consumption of meat.

He is also in favour of the long-continued administration of iodide of sodium in doses of 0·1 to 0·3 gram., the avoidance of acids, and the temporary use of pulv. fol. digital. (0·05 gram.), etc. Further contributions on this subject have recently been made by Erlenmeyer (*D. Medizinalz.*, 1904); Müller-Inada (*D. m. W.*, 1904); Klemperer (*Therap. d. Geg.*, 1905); Mohr (*B. k. W.*, 1906), Minkowski (*Therap. Monatsh.*, 1907), and others.

All authors are agreed that alcohol and coffee, strong spices, and heavy smoking, etc., must be forbidden. Should the conditions favourable to the onset of hæmorrhage be already present, it is specially necessary that everything that leads to a sudden increase of the blood-pressure and causes a determination of blood to the brain should be avoided (see previous chapter).

Improper treatment during the apoplectic attack would have very serious consequences; great caution and mature consideration are therefore required. If it is not definitely certain whether the lesion is hæmorrhage or softening, one must resort to the following measures: the patient must lie quiet in a darkened room, with the head slightly raised and the neck free. If he has fallen to the ground then the clothes must be loosened around the neck and the patient placed in bed with the head well supported, all jerky movements being avoided during the process. The relatives should be warned that the patient will likely waken after a time from this condition, and that he will probably be unable to move the limbs of one half of the body. All disquieting news must be strictly forbidden, and only a member of the family or some careful nurse must be with the patient to guard him as far as possible from any excitement and from any physical exertion. The patient must first of all be forbidden to attempt any active movement, and in particular any movement of the paralysed limbs. Should there be difficulty in swallowing, special care is necessary, while feeding the patient, to avoid setting up coughing.

[1] *Verhandl. d. Kongr. f. inn. Med.*, 1904; *D. m. W.*, 1905. See also ref. in *Therapie d. Geg.*, 1904.

The bowels should be emptied by means of enemata, assisted by the administration of mild purgatives.

If, during the attack, the face is red and bloated, the pulse full and strong, and if from the other conditions a cerebral hæmorrhage has been diagnosed, *venesection* is advisable—especially if the coma last an excessively long time. The blood may be taken from the usual part in the upper arm or from a vein on the dorsum of the foot. It is shown by Wulsten's interesting communication that this procedure sometimes saves life (*D. m. W.*, 1904). It must, however, be avoided if there is general weakness, or especially cardiac insufficiency, when the pulse is small and accelerated, or when other signs points to the process being of the nature of a softening.

While Monakow hesitates to employ venesection, Goldscheider recommends it on the whole, and has recently outlined the indications for its use (*D. m. W.*, 1907): he advises the withdrawal of about 7 to 9 oz. of blood by means of Strauss's canula.

It is unjustifiable to make any attempt to rouse the patient from coma. If stertorous breathing is present he should be placed on his side, and it may be necessary to facilitate the entrance of air by drawing the jaw and tongue forcibly forward.

Traumatic cerebral hæmorrhages have given an occasional opportunity for operative measures—even when the sub-cortical white matter was the seat of hæmorrhage. This method of treatment has been successful in some recorded cases. Although Neisser's cerebral-puncture may give some indication regarding the site of hæmorrhage, I cannot at all recommend operative treatment of spontaneous (non-traumatic) cerebral hæmorrhage.

In cerebral hæmorrhage it is advisable to apply an ice-bag to the half of the skull over the hæmorrhage. Since this procedure, so far as we know, is harmless, it may be safely employed in cerebral softening. If there be cardiac weakness, small quantities of wine or brandy may be administered, also medicinal stimulants such as injections of ether and camphor, coffee, benzoate of soda, etc. In cerebral hæmorrhage the administration of alcohol is altogether prohibited during the early period, but where the patient has been accustomed to its use and the danger of delirium is great, the cautious use of spirits cannot be withheld. Coffee and tea are likewise to be forbidden. Milk, cocoa, lemonade, and non-aerated seltzer water are suitable drinks. During the early days the diet should be principally fluid; after the third day easily digested solid food may be given in small quantities. In all cases the physician must take every precaution to prevent digestive disorder with its consequent vomiting.

If there are no definite indications for treatment no medicines [1] should be administered at first. If the patient be restless the bromide preparations may be employed, and for severe headache or continued sleeplessness an opiate should always be given. Sulphonal, trional, and especially veronal are good in such cases. If there are any grounds for suspecting syphilitic endarteritis, iodide of potassium and mercury should be given: the former may also be prescribed to induce absorption of a hæmorrhage. Widal [2] recommends cyto-diagnosis to ascertain the syphilitic

[1] Wiechowski's (*Arch. f. exp. Path.*, 1905) experimental data deserve consideration on this point.

[2] *Sem. méd.*, 1903.

element, as lymphocytosis of the cerebro-spinal fluid is conclusive evidence of this.

In uræmic hemiplegia lumbar puncture may be of benefit (Wilson, *Journ. of Am. Assoc.*, 1905).

Special care should be taken to avoid bed-sores by means of position and extreme cleanliness. A water cushion ought to be used.

For the *hemiplegia, treatment* must at first be limited to gentle massage of the paralysed limbs, and passive movements of the separate joints—especially those of the arms—to counteract contracture, and the ill effects arising from inactivity. At the commencement five, then ten to twenty minutes (at the most) are sufficient, and should the patient be excited or exhausted by the treatment, the time must be curtailed accordingly. The onset of joint-adhesions and contracture are best obviated by this method. Foester recommends that the position of the paralysed limbs should be frequently altered. Later on the patients themselves may be instructed how to carry out passive movements of the paralysed extremities with the aid of the non-paralysed hand. Lazarus again advises practice in the relaxation of the spastic limbs, *e.g.* the patient must endeavour to voluntarily relax the contracted muscles. It seems to me, however, that these "atonic gymnastics" have no real practical value. Geigel, Lazarus,[1] Konindjy, and particularly Foerster,[2] give very minute details regarding the carrying out of these manipulations.

After two or three weeks, electrical treatment is also advisable. Direct galvanisation to the head must be avoided, but it is permissible to stimulate the muscles of the paralysed extremities by the faradic current. As soon as the tendency to contracture makes itself visible, care must be taken in using electricity to limit the electrical stimulation as far as possible to the antagonists of the contractured muscles. The electrical treatment may be used daily for five minutes for about four or five weeks, and after an interval may be resumed and continued for months. In some cases I have been satisfied of the value of this treatment, *e.g.* in one case the hemiplegic patient was able immediately after treatment to write fairly well and smoothly, whereas before it he could write only with difficulty and indistinctly. Another, who had been paralysed for many years, was able to extend the foot. If hemianæsthesia is marked it is advisable to stimulate the insensitive skin by the faradic brush. Vulpian has noted favourable results from this treatment, as I myself have done. Treatment of the paralysed extremities with the galvanic current is likewise justifiable.

Gymnastic treatment is also employed at this stage with favourable results. It consists at first chiefly of passive movements, and later of voluntary movements.

There is seldom any question of operation, but tenotomy of the Achilles tendon and muscle-transplantation have been frequently carried out in hemiplegia with contracture. Hoffa [3] has described a method of overcoming the excessive action of the pronators and of strengthening the supinators by means of muscle transplantation.

The question then arises, When ought the patient to get up ? As soon as the first movements in the leg set in he desires to attempt to walk, and the physician, in order to satisfy himself of the progress, is rather

[1] *Z. f. phys.*, Th. v.
[2] Abs. *Th. d. G.*, 1904. See also Meige, *R. n.*, 1905; Faure, *R. n.*, 1906.
[3] *Kongr. d. D. Ges. f. Chir.*, 1904 *Cf.* also Bade, Fraenkel, *Z. f. orthop. Chir.*, xv.

inclined to fall in with his patient's wish in this respect. There is nothing to be said against one such trial, but, even in slight cases, this should not be made for three weeks. The patient must on no account be allowed to remain up at this stage.

There can be no question that these attempts at movement encourage the development of contracture. Even after the leg has regained a certain degree of mobility, the patient ought still to spend the greater portion of the day in bed; and it is advisable that the attempt to walk should be preceded for some time by the exercise of walking movements carried out in the lying and sitting positions. On getting up, special care must be taken to support the paralysed arm by a bandage. If cyanosis or a feeling of coldness in the paralysed limbs be present, bandaging has a beneficial effect, but it should not be too firm. Massage and particularly passive movements are good for the joint changes. Liniments may also be used. Subcutaneous injections of strychnine are of no value in hemiplegia.

Erben draws attention to the fact that in certain cases (when the knee cannot be voluntarily flexed) it is advisable to make the patient in walking lead off with the paralysed leg and bring up the sound one, so that the foot does not pass but only reaches the paralysed foot.

Hoffa, Lazarus (*B. k. W.*, 1902). and others have recommended the use of splints in hemiplegic paralysis of the leg, with elastic bands to replace the deficient flexion of the knee and extension of the foot. Alexander has described an apparatus for the prevention of flexion—and pronation-contracture in the arm.

If the apoplectic seizure has left behind it an aphasia, the condition may be improved by suitable education (see p. 742).

In conditions resulting from apoplexy, hydro-therapeutics are as a rule of little use. Hot and cold baths are to be prohibited, only those of lukewarm temperature (26 to 27° R.) being suitable. Even at these temperatures baths must be allowed with great caution, at first only in the form of washing, sitz, or foot baths—and these not before the fifth or sixth week. It may, nevertheless, be helpful to give passive and active gymnastic treatment in a tepid bath. A carefully supervised hydropathic course may be tried during the convalescent stage at Oeynhausen, Wiesbaden, etc. The mild cathartic mineral waters may be recommended, or indeed the mineral water cures themselves (Kissingen, Marienbad, Homburg, etc.). Huchard unjustifiably disapproves of the use of the carbonic acid bath in arterio-sclerosis. The whole mode of life must be regulated in such a way that all determination of blood to the head is avoided (*cf.* chapter on cerebral hyperæmia).

Cerebral Softening (Encephalomalacia)

The usual *cause* of cerebral softening is local anæmia of the brain substance brought about by blocking an artery. The portion of the brain deprived of nourishment undergoes a process of degeneration which is manifested by an actual *softening*—in the original sense of the term. This occlusion of vessels is *embolic* or *thrombotic* in nature. The embolus usually originates in the heart (from a valvular lesion—especially mitral stenosis, or cardiac failure with thrombus-formation), less frequently in the aorta (atheroma aneurism), and still more rarely in the pulmonary veins (in ulcerative bronchitis, cavity-formation, and gangrene of the lungs).

In very rare cases particles of tumour from the heart or lungs reach the brain. The embolism may also consist of other cellular elements, but it is unnecessary here to consider these unusual occurrences—amongst which, for instance, may be reckoned the so-called echinococci emboli. More frequently micro-organisms are the elements conveyed by the embolus to the brain.

The embolus may also be a product of the disintegration of a thrombus that has settled in one of the larger cerebral arteries.

Thrombosis of the cerebral vessels is rarely developed except through disease of their walls, and usually in consequence of ordinary *senile atheroma*, *i.e.* arterio-sclerosis (Marchand) or *specific endarteritis*. A very similar condition of the cerebral arteries may, however, be produced by chronic intoxication (alcohol, lead, etc.), or may be inherited. Further, it is my experience [1] that diseases of the nervous system, which are associated with continued disturbance in the function of the innervation of the heart and vaso-motor system, such as cardiac neurasthenia and certain forms of traumatic neuroses, may be the exciting cause of the development of atheroma at an earlier period of life. This fact, on which I have long laid stress, is now almost universally acknowledged. Continued and powerful mental emotion may also produce the same effects.

Cranial injuries are also frequently followed by premature disease of the vessels of the brain (Friedmann,[2] Oppenheim,[3] Kronthal-Sperling,[4] Windscheid,[5] and others). Sänger and Windsheid, amongst other investigators, have found that arterio-sclerosis is very common amongst the working classes. This is due to bodily exertion, the abuse of spirituous liquors, and other causes.

It is assumed that certain diseases increase the coagulability of the blood and may thus cause thrombosis, even although the vessel wall is completely intact. To this category belong acute infective diseases, phthisis, and puerperal fever.[6] Typhoid hemiplegia can frequently be traced to thrombosis (W. Osler). Many writers hold that the presence of micro-organisms and poisons in the blood raises its coagulability. Others are of opinion that an endocarditis is first produced by the infective disease; this is followed by the development of an embolus, which becomes manifest in the form of a thrombosis, after the endocarditis has healed, or even if it remain latent. In most of the cases of post-diphtheritic hemiplegia submitted to post-mortem examination emboli have been proved to be the cause (Slawyk,[7] Baginsky[8]). In two cases of parametritis, I have noticed complications which I could only ascribe to an embolic or thrombotic closure of the artery of the Sylvian fissure. The production of thrombosis or embolism is favoured by great cardiac weakness, as that is the chief cause of the formation of clots. Slowing of the circulation favours the development of thrombosis but can hardly of itself produce it. If, however, the coagulability of the blood has been increased by any of the already-mentioned causes, a thrombosis may form either where the blood circulation is slow, or where eddies are formed in it (Recklinghausen), or where it passes from a narrow to a wider lumen. *Chlorosis* and *leukæmia* also favour the production of thrombosis.

[1] See the first edition of "Traumatischen Neurosen," Berlin, 1889, p. 120, and other parts of my writings.

[2] *A. f. P.*, xxiii. [3] *L. c.* [4] *N. C.*, 1889.

[5] *M. m. W.*, 1902.

[6] On the cerebral paralysis appearing in pregnancy and the puerperium consult the works of Hösslin (*M. m. W.*, 1904, and *A. f. P.*, Bd. xxxviii.), which also give the literature on this subject.

[7] *Charité-Annalen*, xxiii.

[8] "Lehrb. d. Kinderkr. und Nothnagels Handbuch," ii.

In cases of CO-poisoning softenings occur, the origin of which has not yet been fully cleared up. The softenings are specially liable to affect the lenticular nucleus on both sides (Poelchen,[1] Kolisko). Even after extensive cutaneous burns, multiple thrombosis could be recognised in the brain (Klebs). We can here merely refer to traumatic encephalomalacia (*cf.* remarks on traumatic secondary apoplexy, pp. 745 and 759), softenings in the neighbourhood of hæmorrhages and tumours, and the rare phenomenon of thrombosis in tubercular meningitis, which may also affect the veins.

Nor can we discuss in this place the other diseases, hyaline, cartilaginous (Marburg), of the cerebral vessels and the endophlebitis, which is usually of syphilitic origin (Rieder, Bartels, and others).

When the conditions necessary for the production of embolism or thrombosis are already present, any mental agitation may act as the exciting agent. Fear, in particular physical over-exertion or labour, are important—the latter especially in the production of embolism. There have been a few observations on the development of a hemiplegia during *narcosis* (Schwartz,[2] Büdinger,[3] Chipault), without its being possible to ascertain definitely whether hæmorrhage or softening was the actual cause.

Emboli remain fixed, usually at the point of division of one of the larger arteries, most frequently in the arteries of the Sylvian fissure and their branches—preferably the left side. The internal carotid artery and the posterior cerebral artery are also not infrequently the seat of emboli, and sometimes the vertebral arteries, especially the left. Thrombi may develop at any point, but they usually form in the large vessels at the base of the brain, most frequently in the arteries of the Sylvian fissure, the internal carotid, basilar, and posterior cerebral arteries or their branches.

The embolus, which forms a firm, colourless clot (occasionally calcareous), adhering to the vessel wall, may cause a secondary thrombosis in the neighbouring branches of the vessel. These secondary thrombi are red and loose. In fact, the thrombus frequently spreads in this manner and obliterates the branches on the distal side of the obstruction in the vessel. Sometimes the embolus becomes disintegrated before it has caused destruction of tissue, and the resulting particles are carried away in the blood-current. This is specially the case with emboli which contain specific material, *e.g.* micro-organisms. The thrombus can also be reabsorbed.

In specific endarteritis which attacks principally the larger branches of the circle of Willis, there may be occlusion of the vessel lumen over a considerable distance.

The occlusion of the vessel lumen only causes softening in the corresponding area of the brain in the absence of the conditions necessary for the establishment of collateral circulation. This is the case when the arteries are end-arteries, such as those which supply the white substance of the brain, or when the vessel is so extensively occluded that the origin of the lateral branches which effect the collateral circulation is also blocked within the main vessel. Thus, it happens, for example, that thrombosis of the internal carotid, which usually extends to the origin of the anterior and middle cerebral arteries, has very serious and permanent

[1] *V. A.*, Bd. cxii. See also Sibelius, *Z. f. K. M.*, Bd. xlix., and *M. f. P.*, xviii; Ergänz, Knecht, *D. m. W.*, 1904.

[2] *Gaz. des hôp.*, 1897.

[3] *A. f. klin. Chir.*, Bd. xlvii.

results, while both after ligature or in embolism collateral circulation may be re-established through the circle of Willis. Collateral supply is more easily established in the brain cortex than in occlusion of the arteries of the interior of the brain. Thus, even when the artery of the Sylvian fissure is blocked, softening of the *cortical area* may be absent or very limited, whereas the *white matter* supplied by it is always very extensively softened.

Monakow states that if occlusion of the artery of the Sylvian fissure takes place before the origin of the basal arteries, which penetrate to the interior of the brain, the whole lenticular nucleus, the anterior part of the optic thalamus, the sub-thalamic region, the central convolutions (for the most part), the island of Reil, the operculum, and the third frontal convolution will be destroyed. When the posterior cerebral artery is occluded, the greater part of the occipital lobe, especially the calcarine fissure and the cuneus, also the posterior part of the optic thalamus are cut off from the blood circulation, but the actual extent of the softening depends mainly on the site of the clot and the establishment of collateral circulation.

Foci of softening may be found in all parts of the brain and are only apparently more frequent in the cortex because of its wide extent. They also often develop in the deep layers of the centrum semiovale. The head of the nucleus caudatus and the anterior part of the putamen are also favourite sites. They are very rarely found in the cerebellum.

The process of softening does not take place immediately after the closure of the vessel. Thirty-six to forty-eight hours, and in exceptional cases three to four days may elapse before disintegration and diminution in the consistency of the brain tissue set in. Swelling and serous infiltration of the affected area, however, immediately follow the embolism.

Should the vessel lumen not be completely occluded no further changes may occur, or necrotic (and indurative ?) processes may arise which do not lead to complete softening. This has been assumed to be the origin of certain forms of lobar sclerosis. I have the brain of a case observed by Schönfeldt (Riga)—that of an adult in whom diffuse sclerosis and atrophy had developed in this manner over a whole hemisphere. Moreover, a simple œdema or white softening of the brain has also been met with in cases in which the lumen of the carotid has been gradually narrowed through obliterative arteritis.

Red, *yellow*, and *white* softening are described. The colour depends in the first instance on the amount of blood present. Red softening is observed specially in the cortex because the grey matter contains more blood. It occurs also in the central grey matter, especially near the aqueduct of Sylvius. In a few weeks the red softening—which always represents an early stage—becomes yellow, and forms the yellow patches (plaques jaunes) of the brain cortex. This is brought about by advanced fatty degeneration of the diseased tissue and especially by the transformation of the colouring matter of the blood into pigment. The development of an actual hæmorrhagic infarct in the sense of Cohnheim is most unusual in the brain (according to Schmaus this never occurs), but it is quite common to find small extravasations of blood in the periphery of the softened area. The latter has in the white matter usually a white or whitish-blue tinge. The tissue is of the consistence of pulp or is even completely liquefied (Schmaus speaks of colliquation necrosis), and has the appearance of milk, of lime. It contains myelin drops, detritus, and very numerous granular cells, which distinguish with certainty true softening from post-mortem maceration.

After the absorption of the liquid material cysts may form, but the

centre of the softening may, on the other hand, continue for years in its original form and condition. Cortical softenings usually result in cicatrisation. In the neighbourhood of small arteries a circumscribed perivascular sclerosis is frequently to be found instead of softening.

For the histo-pathology of these processes see Saltikow, *A. f.*, Bd. xl., xli.

The size of the areas of softening varies from that of a pin-head, or even microscopic minuteness, to that of a fist. It may indeed occupy the greater part of a hemisphere, *e.g.* in thrombosis of the carotid. I have found in one case the whole hemisphere transformed into an area of softening. In atheroma, instead of one large centre or at least in addition to it, are found numerous smaller centres—some of which can only be discerned microscopically. It is also by no means uncommon to find bilateral centres symmetrically distributed in both hemispheres, *e.g.* in both lenticular nuclei and in both occipital lobes.

Embolism occurs most frequently in youth and middle life, whereas thrombosis, if we except syphilitic cases, is to be found almost exclusively in the aged.

P. Marie [1] frequently found as the essential change in senile hemiplegia a *lacunar* condition in the brain, especially in the region of the lenticular nucleus.

Symptomatology.—Cerebral softening, like cerebral hæmorrhage, is characterised by *transient* and *permanent*, *general* and *local* symptoms. In larger areas of softening, especially those of embolic origin, an attack of apoplexy is the first symptom. There may be embolism of the smaller branches and thrombosis of even the larger arterial trunks without mental deterioration. The coma is, as a rule, not so profound, and is of shorter duration than that which accompanies cerebral hæmorrhage. In both a fall of temperature is usually absent, while eight to ten hours after the illness commences a rise in the temperature as a rule begins—not indeed a serious one, but lasting perhaps for several days. Sometimes fever sets in only after several days have elapsed. A greater rise of temperature and rigors occur only when the embolus is septic.

Embolism of a large artery is almost invariably accompanied by *loss of consciousness*. The cause of this is the sudden disturbance to the circulation, not by any means limited to the area supplied by the vessel affected, but extending more or less to the whole brain. This is accompanied by a kind of shock (Trousseau, Wernicke), *e.g.* a mechanical injury to the brain causing the vessels, emptied of blood, to collapse and thus bringing about a local displacement of the surrounding tissues, etc. In addition to this, we agree with Monakow in considering it probable that the mechanical stimulus affects the vaso-motor centres reflexly and in

[1] *Revue de Méd.*, 1906. Ferrand ("Essai sur l'hémiplégié des vieillards," Paris, 1902) has—on the strength of his own investigations—made a very exhaustive criticism upon Marie's statements. Dupré and Devaux also have discussed the question. The small cavities are found most frequently in the central ganglia. They arise from necrobiotic, sclerotic, and encephalitic processes in the immediate neighbourhood of arteries. Pick has ascribed them to lymph-stasis. Schröder has very severely criticised these interpretations, and assumes on the one hand that the usual process of softening is going on, and on the other hand that special cavities arise on account of the general senile shrinking of the brain tissue together with the immersion in hardening fluids. Against this, however, is the fact that Marie is well acquainted with the artificial cavities produced by gas-formation, and describes them with Guillain as "Porose cérébrale." The question is also discussed by Grasset (*Sem. méd.*, 1904), Catola (*Revue de Méd.*, 1904), Lévi ("Le Cerveau sénile," *R. n.*, 1906, and Disc.), and by Bechterew (*M. f. P.*, xiv.).

this way produces a general cerebral anæmia. An epileptic attack—either general or partial—or even a form of " status epilepticus " may be present in place of the coma. Vomiting is also not unusual. More rarely embolism manifests itself in the first instance only by a slight attack which, later, passes into a condition of stupor with delirium—phenomena which one is inclined to ascribe to the reactive processes going on in the neighbourhood. Thrombosis of the larger arteries is also ushered in by an apoplectic attack, but this is more frequently absent than in cases of hæmorrhage, or the disturbance of consciousness is only slight. It also frequently occurs in thrombosis that the signs of paralysis precede the disturbance of consciousness and that the latter is exchanged for a state of mental confusion, or slight delirium extending over several days. If there is no stroke, as is usually the case in occlusion of the small arteries, and is even more frequent in thrombosis, the patient complains of headache and giddiness. Here also *premonitory symptoms* play a much more important part than in cerebral hæmorrhage. It is true that embolism develops suddenly without being preceded by any brain symptoms, but thrombosis is preceded for days, weeks, and even years by certain complaints which indicate cerebral disease. The general consequences of atheroma or specific endarteritis are headache—which is usually intensified by coughing, straining, and by the dependent position of the head, giddiness, frequently also failure of memory and of intelligence, states of mental confusion and excitement often combined with hallucinations, and especially a lack of the sense of localisation, both in respect of time and place, sometimes also repeated states of slight stupor which are the expression of transient disturbances of the circulation or occasioned already by smaller centres of softening, or an intermittent dysarthria or aphasia.

Arterio-sclerosis in very rare cases leads to an affection of the optic nerve demonstrable ophthalmoscopically (see p. 714), whereas a concentric diminution of the field of vision frequently occurs (Vogt). Further, the pressure of the dilated vessels, with their rigid walls, on the neighbouring parts of the brain, especially on the pons and medulla oblongata as well as on the basal cranial nerves, especially the oculo-motor nerves, produces various symptoms, as has been specially pointed out by Oppenheim and Siemerling (*Charité-Annalen*, xii.), (compare the chapter on compression-bulbar-paralysis and pseudo-bulbar-paralysis). Elliott Smith (*R. of N.*, 1905), who made the same observations, seems ignorant of our work. We cannot here discuss the symptoms arising from the arterio-sclerosis often co-existent in the heart, the aorta, the kidneys, the intestines. See p. 675 for the Adams-Stokes group of symptoms.

Infarction of some other organ (kidney, spleen, etc.) may precede the embolus. The combination of a cerebral embolus with one of the central artery of the retina is particularly interesting. The resulting symptoms are usually those of the visual disturbance combined with hemiplegia of the opposite side.

The typical form of the paralysis caused by encephalomalacia is *hemiplegia*, which appears in exactly the same way as in cases of cerebral hæmorrhage (compare for its symptomatology, p. 684, and the previous chapter). It is only now and then, in senile atheroma, and more frequently in specific arteritis, that we have a development of paralytic symptoms occurring in such a way that hemiparesis precedes hemiplegia, or that one limb and, after one or two hours or even on the following day, the other is affected, or that unilateral paræsthesia precedes the onset of paralysis and hemianæsthesia.

Hemiplegia, when affecting the right side, is frequently accompanied by *aphasia*, and this is mostly a direct focal symptom. As areas of softening are not infrequently limited to certain circumscribed cortical areas, *monoplegia* with aphasia, or *aphasia* by itself, or *hemianopsia*, are often the only persistent symptoms of this cerebral affection. As, however, softening also produces *indirect* symptoms, and the occlusion of the vessel-lumen is particularly liable to affect the middle cerebral artery, it is easily conceivable that hemiplegia—alone or combined with the above-mentioned symptoms—is the most common sign of cerebral softening. If this focal symptom is an indirect one, *i.e.* if the focus of softening does not directly destroy the motor path or the motor centres, it soon disappears. Hemiplegia may be accompanied by hemianæsthesia, or the latter may alone be present or may be accompanied by hemiataxy.

The term *transient hemiplegia* is used when softening does not result—either because the clot dissolves or is washed away, because collateral circulation is established, or because (as in syphilis) the defective blood supply was the result only of great narrowing of the vessel-lumen, which, for the time being, amounted to occlusion. This hemiplegia disappears within a few hours or days.

As foci of softening may develop at any part of the brain, there is no cerebral symptom which may not have this as its cause.

At times the outward manifestations do not correspond with the site of the area of softening; they defy the law of localisation. We must always, however, remember that in addition to the large focus, numerous small ones are often distributed throughout the brain—many of which can be discovered only microscopically. Cases of this kind have been communicated both by Siemerling and myself.

Symptoms of occlusion of the *internal carotid*. If collateral circulation is established the hemiplegia diminishes rapidly or is quite absent—as in the case of ligature. If the vessels are too narrow to admit of a sufficient supply of blood, or if they are lacking altogether, or if the thrombus extends to the anterior cerebral or middle cerebral artery, a very extensive area of softening is formed; this gives rise to hemiplegia with deep and persistent coma and usually to a rapid fatal termination. A more or less serious loss of sight may be caused by occlusion of the ophthalmic artery (and central artery of the retina). In the case of embolism of the carotid the symptoms vary according to the precise site at which the clot is situated.

A thesis by Lestelle (Paris, 1903) is a comprehensive and recent study of the phenomena which follow ligature of the carotid.

Occlusion of the main trunk of the *middle cerebral artery* causes complete hemiplegia with transient or persistent hemianæsthesia, and, in the case of occlusion of the artery on the left side, complete or partial aphasia. If the occlusion is limited to one of the branches the symptoms, as will be easily understood, are modified according as the consequent softening occupies the third frontal convolution, the parietal lobe, or the first temporal convolution. If it is a case of embolism and the thrombus has extended into the cortical branches of the central convolutions, unilateral convulsions sometimes precede the attack of paralysis. According to the extent of the lesion, a monoplegia (facio-lingual, facio-brachial,

etc.), a hemiplegia, or a motor aphasia, with or without paralysis, will develop. Thus, occlusion of branch I. of the left side is accompanied by motor aphasia with monoplegia (right facio-lingual), and the arm may at the same time be paretic. The obstruction of branch II. is followed by hemiplegia or monoplegia (facio-brachial); that of III. or IV., which supply the lower part of the parietal lobe, if on the left side, would result in alexia, usually also hemianopsia, and even auditory aphasia. We should expect the last to be the main symptom in occlusion of branch V. For the symptoms that follow softening of the upper part of the parietal lobe, *cf.* pp. 626 and 627, and 704 *et seq.*

Extensive or multiple smaller foci of softening have been observed in the area supplied by the artery of the Sylvian fissure, and in these cases the clinical phenomena have been strikingly insignificant as compared with the extent of the pathological process (Marie, Bikeles).

Embolism or thrombosis of the *anterior cerebral artery* (the former is seldom found) would result in a crural monoplegia and psychic disturbances (?).

Occulsion of the branches which penetrate to the white matter usually produces hemiplegia (or hemianæsthesia); if, however, the softening does not extend beyond the region of the caudate and lenticular nuclei, or the optic thalamus, hemiplegia may not appear at all.

The symptomatology of softening, limited to the central ganglia, requires further investigation. Unilateral softening of this region, if circumscribed, may not give rise to any symptoms. On the other hand in these focal lesions symptoms of motor irritation of different kinds (*cf.* pp. 690 *et seq.*), signs of sensory irritation, reflex phenomena, and, especially in the case of bilateral foci, difficult articulation, or deglutition, hypermimia, rarely amimia (or loss of power to communicate thought by gesture), and urinary disturbances, etc., have been observed. See also chapter on pseudo-bulbar paralysis. The partial paralysis of the limbs may, under these circumstances, be a non-spastic one (normal toe-reflexes, etc.).

Occlusion of the *posterior cerebral arteries*—if collateral circulation does not set in—is usually followed by hemianæsthesia, and invariably by hemianopsia.

Of thrombosis of this artery Monakow, whose knowledge of the pathology of this region is most intimate, sketches the following picture: If the thrombosis set in gradually, as it usually does, there first appear for a time general phenomena, such as attacks of giddiness, states of forgetfulness, transient hemianopic disturbances, entoptic scotoma with headaches and transient dimness of vision; there follows a regular apoplectic seizure with resulting hemiplegia, sometimes also with convulsive movements. When the general symptoms have passed off, the hemiplegia usually also disappears and *hemianopsia* now becomes the most prominent, and, if the thrombosis is on the left side, there is sensory aphasia, *i.e.* paraphasia, and amnesic aphasia. Hemianasthesia may also be present.

Regarding the symptoms which appear in thrombosis of the basilar and vertebral arteries and their branches, especially the cerebellar arteries, see the chapter on acute bulbar paralysis.

There are certain cases of arterio-sclerosis in which a number of smaller foci of softening develop without any actual attack of apoplexy, and the paralytic symptoms have a more or less cumulative course, so that in their development and progress they correspond rather to the type of a chronic disease. In the long run as a rule there is disturbance of the mental equilibrium and frequently convulsive outbreaks of laughter and weeping, and bulbar symptoms are not uncommon (*cf.* chapter: Pseudo-bulbar-

paralysis [1]). True attacks may also occur at any time with typical signs of paralysis. In this *multiple softening* a form of amnesia sometimes occurs which may simulate amnesic aphasia without any corresponding affection of the speech centres. The condition has a close resemblance to senile dementia and often passes into it. It is, however, also possible to have a diminution of mental power, associated with a *single* apoplectic seizure, which has led to hemiplegia. This condition has all the appearance of a pronounced dementia (dementia apoplectica, dementia post-apoplexiam, post-hemiplegic dementia). Such cases are mostly due to diffuse disease of the cerebral arteries and the conditions to which they give rise—Mingazzini calls attention to this, also (*Riv. sper. di Fren.*, 1897) —and very often to syphilis. This dementia is therefore very seldom the consequence of an apoplectic stroke, nor is it caused merely by the lesion which this has occasioned, but it is due rather to its combination with a general disease of the cerebral vessels and their consequences.

The essential points relating to arterio-sclerotic dementia are discussed in the treatise of Oppenheim and Siemerling (*Charité-Annalen*, xii., and *B. k. W.*, 1887), but are largely neglected by later writers. These conditions have also been specially alluded to by Binswanger, Alzheimer (*Z. f. P.*, Bd. lvi., lvii., and lix.), Buchholz (*A. f. P.*, Bd. xxxix.), Barret (*Amer. Journ. of Insanity*, Bd. lxii.), Weber (*M. f. P.*, xxiii., Ergänz), and Campbell (*R. of N.*, 1907).

Diffuse atheroma of the brain with the resulting pathological changes are associated with other phenomena which are very difficult to interpret—especially various disturbances of the gait, which may partly resemble that of paralysis agitans and partly remind us of basophobia. The patient remains standing in a nervous state, cannot take a step forward, or moves on the same spot, leans or walks backwards, etc.; but, when one leads him a few yards or has even energetically encouraged him by word, he can walk a considerable distance quite well or at least with a sort of tripping step. Petrén (*A. f. P.*, Bd. xxxiii. and xxxiv.) has carefully described these phenomena, and I myself, long ago, made similar observations. Pelnar also treats of them. Petrén's idea is that we have a combination of an organic disease of the nervous system with a functional one, and he attributes the locomotor disturbance to the latter. It appeared to me that these peculiarities of movement were partly the result of injury to the centres for equilibrium, which constitute the co-ordinating apparatus for the movement of walking, and partly a result of the dementia itself, and both of these factors might be traced without hesitation to the process of atheroma. Naunyn (*Volkm. Saml. N. F. Nr.*, 391, 1905) also treats of this question. He refers the ataxia to the general injury to the brain from the arterio-sclerotic process, and looks upon it as the special reaction of old age to these changes.

Differential Diagnosis.—To decide between softening and cerebral hæmorrhage the signs already mentioned (p. 806) are of value. In addition it may be mentioned that during the apoplectic attack from softening the face is neither puffed out nor red. If the objective phenomena point to a cortical area (aphasia, monoplegia), softening will probably be the cause.

It may be difficult to distinguish between cerebral softening and tumour if the former is preceded by long-continued premonitory symptoms, or if, on the other hand, the tumour after remaining latent for a time suddenly produces signs of paralysis. In cerebral softening, however, the typical signs of increased cerebral pressure are absent: the choked disc, the slowed pulse, and the gradually increasing stupor. In cases where the slowing of the pulse is caused by atheroma of the coronary arteries

[1] This description—already given in the second and third editions—corresponds in most points to the data given by Marie-Ferrand in connection with the symptomatology of their lacunar porosis and the "Hémiplégie des vieillards." They lay special stress on the incompleteness and transitory character of the hemiplegia and the non-appearance of contracture. Refer also to the exhaustive study of Jacobsohn (*A. f. P.*, Bd. xxvii. and xxviii.).

complicating the condition, this reveals itself by other signs. It must be remembered, however, that the presence of atheroma by no means excludes the possibility of a cerebral tumour. Vomiting rarely occurs in thrombosis except at the beginning: an exception to this is thrombosis of the basilar artery, which, however, may be recognized by other significant signs. The convulsions occasionally set up by embolism and thrombosis, even when unilateral, do not display the regular course of cortical epilepsy.

A short time ago I was called to a case in which the combination of unilateral optic atrophy and symptoms of paralysis on the opposite side of the body, together with the presence of severe localised headache and corresponding tenderness of the skull (also the dulling of the percussion-resonance at this spot), led me to the mistaken diagnosis of a tumour. The autopsy, however, revealed an immense area of softening, together with a local accumulation of fluid at the spot where there had been pain and tenderness. The facts that intermittent claudication had preceded and that symptoms of heart-trouble accompanied the disease should have led to a correct diagnosis.

The determining factors for a differential diagnosis in the case of an abscess on the brain must be looked for in the corresponding section.

The localised atrophy of the brain which occurs in very old people may —according to the observations of A. Pick,[1] Liepmann,[2] Mingazzini, Stransky,[3] A. Westphal, and others—present a group of symptoms similar to that of softening, but it lacks the signs of an apoplectic stroke; also the objective symptoms arise gradually—just as in progressive softening (see Appendix)—and the symptoms of dementia senilis are usually present, which fact is specially decisive in the diagnosis.

The mistake is specially apt to arise in cases where softening shows itself—not by focal symptoms, but by general cerebral symptoms such as intracranial pressure, giddiness, stupor, sleeplessness, etc.—of confounding it with a neurosis, especially with hysteria and neurasthenia. The persistence of the symptoms, the fact that they are not entirely subjective, the signs of vessel disease, and a stupor appearing objectively, all point to softening. Moreover, an area of softening of any considerable extent tends with time to injure the general health and even to cause marasmus.

For " acute senile encephalitis " see the next chapter.

Burian (*Casop. ces lék.*, 1904) describes an interesting but obscure case of thrombosis of the vena magna Galeni.

Finally, it must be noted that there are cases of hemiplegia in which, post-mortem, no changes can be found in the brain. This has been confirmed in *tuberculosis*, *alcoholism*, *uræmia*, *diabetes*, *lead-poisoning*, *arteritis*, and *pneumonia*. It is sometimes specially difficult to distinguish a uræmic hemiplegia from that caused by cerebral hæmorrhage in a case of contracted kidney. From the numerous observations of others (Lancereaux, Rosenstein, Baginski, Dunin, Senator, Paetsch, Rose, Herzog, Donetti, Ewing, Brodier, and others) and from my own experience it is evident that aphasia, hemiplegia, amaurosis, and other phenomena may be the direct result of the uræmia without any demonstrable local disease of the brain. On the other hand these symptoms, when they arise in the course of uræmia, may be caused by capillary and more extensive apoplexy. If they are merely transitory it is simplest to take for granted that there

[1] *W. kl. W.*, 1901; *M. f. P.*, xix. and elsewhere. [2] *N. C.*, 1900. [3] *Jahr. f. P.*, xxv.

are no organic changes. I[1] had an opportunity of proving this in a case of *carcinomatous cachexia* and gave it as my opinion that it was a question of *toxic focal disease*. Later, Bettelheim[2] published a similar case. Saenger,[3] who found cancer cells in the pia mater, in a case of paralysis with macroscopically negative findings, during the course of a cancerous disease, has contested my opinion, whereas Nonne,[4] who has seen several similar cases, agrees with it. Hemiplegia, without anatomical changes, has also been discussed by Jacobson,[5] who is evidently ignorant of my work. Nevertheless, as far as I can see, there are no striking clinical landmarks to distinguish hemiplegia *sine materia* from the hemiplegia which is caused by organic disease.

The minuter cell-changes described by Weisenburg (*Journ. of Nerv.*, 1904) in uræmic hemiplegia cannot be taken as distinctive. The question has been dealt with experimentally by Dopter (*Arch. de Méd. expér.*, 1903). With reference to the albuminuric paralysis of pregnancy, see Hösslin, *l.c.*

The interpretation of transitory hemiplegias, which occasionally occur in cardiac disease, and which on account of their rapid disappearance cannot be satisfactorily explained by embolism, is still uncertain (Achard and Levi, Marty, Achard-Ramond, ref. *R. n.*, 1905). The same may be said of paralytic conditions, with or without convulsions, which have been frequently observed in diseases of the pleura, especially in empyema and surgical interference with the pleural sac (puncture, incision, irrigation). The phenomenon has been explained as a reflex-paralysis, or "hystero-traumatic paralysis," and has also been referred to embolic processes (Rendu, Jeanselme, Janeway, Cestan). During pneumonia also, a hemiplegia which cannot be traced to local disease may develop. Sometimes cerebral œdema or slight meningeal modifications could be found; at other times there was no pathological condition present or the rapid retrogression excluded the possibility of any gross anatomical change, so that here again we are forced to assume a local action of the infective agent or of its toxine. Of course it is quite possible that a local disease may be the cause of a hemiplegia, arising during pneumonia, as in a case observed by Vogelius. Hemiplegia occurring in a child with intestinal worms (?) is described by Sigaud (*Gaz. des hôp.*, 1904).

Little is known also regarding the cause of *diabetic* hemiplegia, but observation of cases in which the paralysis was only temporary suggests that it may possibly be due to auto-intoxication, perhaps also to disorders of the circulation. I have specially often observed *bulbar symptoms*—transitory as well as permanent ones. In one case of this kind the paralysis first appeared in the eye muscles and bulbar nerves, and lasted from a few minutes to half-an-hour; then followed a seizure, with all the characters of an acute bulbar paralysis, and this disappeared almost entirely in a few days. A short time ago I saw a case of this kind with ophthalmoplegia, partial aphasia, and agraphia. Cerebral focal symptoms, without anatomical changes, have been described also by Redlich (*W. kl. W.*, 1893) and Naunyn (Nothnagel's "Handbuch," vii.). Lépine and Blanc (*Revue de Méd.*, 1886) have noted changes recognisable by the microscope. Great care at the same time should be taken in the prognosis, as the arterio-sclerosis, which frequently exists, may lead to very serious consequences—*e.g.* hæmorrhage in a case given by Klippel-Jarvis (*R. n.*, 1901), and softening by Steinthal, Ferichs, Ogle, and others.

The *prognosis quoad vitam* is on the whole a favourable one—certainly more favourable than in cerebral hæmorrhage. Thus the statistics given by Jones (*Br.*, 1905) show that whereas in cerebral hæmorrhage death ensues in 30 per cent. of the cases within twenty-four hours, in thrombosis it occurs in only 15 per cent., and in cases of embolism in only 7·5 per cent. It is only thrombosis of the basilar and of the carotid arteries that form a real danger to life. A long duration of the loss of consciousness is by no means so ominous a symptom in softening; even when it has lasted five or six days the patient may still recover. But deep coma

[1] *Charité-Annalen*, xiii., 1888. [2] *W. med. Bl.*, 1901. [3] *N. C.*, 1900 and 1901.
[4] *N. C.*, 1900. [5] *Hospit. Tid.*, 1893.

is an unfavourable sign in cerebral softening. In the prognosis the general condition and the state of the heart must be taken into account. If the area of softening is not very large, life may continue many years—even for a period of ten years. On the other hand, I have seen a few cases where the first attack was so slight and transient that it did not come under treatment at all, and the prognosis, considering the absence of symptoms, seemed favourable, whereas a very few days or weeks later a second attack terminated fatally.

The prognosis in regard to the paralytic symptoms is, on the whole, an unfavourable one. If there is not a return to the normal or almost normal within the first two or three weeks, an adjustment can no longer be expected, as collateral circulation is established in a few days and the so-called indirect focal symptoms disappear in a period of from two to three weeks. On the whole the latter do not play such a great rôle in softening as we must ascribe to them in hæmorrhage. Moreover, softening which has resulted from syphilitic endarteritis cannot be recovered from. The prognosis is besides dependent on the site and the extent of the softening.

Treatment.—Blood-letting is quite out of place here, although some physicians still recommend it. During the attack the same precautionary measures, especially in regard to position and rest, should be used, as in cerebral hæmorrhage. If the heart's action is feeble, stimulants should be administered.

If the softening is due to syphilis, potassium iodide and mercury should be at once prescribed. If this treatment is insufficient to remove the softening, the disease of the vessels may yet be arrested by it. Lumbar puncture, with its proof of a lymphocytosis, or Wassermann's reaction, may show that syphilis is the cause.

Regarding the treatment of the paralytic symptoms and the general dietary regimen, nothing fresh need be added to the statements in the previous chapter.

So-called Chronic Progressive Softening of the Brain

A small number of cases have been published which tend to show that softening of the brain substance may develop slowly and progressively. Larger or smaller areas of softening have been found in the white matter of the hemispheres without its being possible to prove that there was any obstruction of the arteries supplying them. The symptoms usually were those of a hemiplegia developing gradually or by successive stages. General symptoms were entirely wanting or were limited to headache. Sometimes local twitchings of the muscles preceded the onset of the paralysis. Sensory disturbances, such as pain and anæsthesia, were also present. The hemiplegia developed usually within a period of months or its progress may sometimes have extended over an even longer time: a period of quiescence then might follow and death result from an intercurrent affection. The patients were almost all of advanced age. In one case of this kind, which I had the opportunity of examining, I found the cerebral arteries free, but the carotid at the neck was thrombosed or closed by an obliterative endarteritis. Brissaud, Massary, and Trénel have described similar cases after I had done so (compare the first edition of this manual, 1894). In the case described by Brissaud-Massary (*R. n.*, 1898), there was an annular endarteritis of the carotid, with a considerable narrowing of its lumen. As for the process in the brain, that had more the nature of an œdema than that of softening.

Henneberg (*A. f. P.*, Bd. xxxviii.) has recently described a case of chronic or subacute softening occurring in the puerperium, in which this could not be traced to vascular disease. Henneberg deduced from the presence of a co-existing myelitis, that there is a softening caused by toxins, which corresponds to the myelitis with this origin. It would appear that chronic forms of encephalitis (see next chapter) occur, which, in their histological characters, are related to simple softening.

Ziehen, from the progressive development of hemiplegia in one of his patients who was suffering from putrid bronchitis, diagnosed a pulmonary abscess and decided to proceed to operation on this assumption; it turned out that there was an area of softening caused by progressive thrombosis. He described several cases of this kind lately (*Med. Klinik.*, 1906). On the other hand, Gombault and Halbron (*R. n.*, 1903) made the mistake of assuming a chronic softening in their case of cerebral tumour on the ground of the progressive development of hemiplegia. See further Hunt, ref. *R. n.*, 1907. Buzzard and Barnes (*R. of N.*, 1906) contribute an interesting observation on a case of bilateral progressive hemiplegia in arterio-sclerosis; they ascribe the process of degeneration to deficient blood-supply in the area of the middle cerebral artery.

We must note here also that form of progressive hemiplegia described by Mills and Spiller (*Journ. of Nerv. Dis.*, 1903; *Journ. of Amer. Med. Assoc.*, 1906; *Univ. of Penn.*, 1906) and referred to a unilateral pyramidal degeneration. We have to deal in this case with the symptoms of a very slowly developing spastic hemiplegia, in which all the complicating symptoms of cerebral disease are wanting. We have observed two cases of this remarkable condition.

The connection between diffuse atrophy of the hemispheres and the restriction of the blood-supply, owing to narrowing of the vessels, as assumed by Gowers, Bailey (*Br.*, 1900), and others, requires further elucidation.

Encephalitis

(*Acute, non-purulent form*)

For literature on this subject see Oppenheim and Cassirer, "Encephalitis," 2nd Edition; Nothnagel's "Handbuch," Wien, 1907.

The term encephalitis is applied to very various forms of inflammation of the brain, *e.g.* the focal change in the brain in multiple sclerosis may be termed a disseminated encephalitis, and further, in the different forms of meningitis (traumatic, tuberculous, epidemic, and pyæmic), alterations of the brain substance occur—especially in the neighbourhood of the affected meninges—which must be placed in the category of encephalitis. Pathological conditions of this kind are also found in *rabies* and *chronic chorea*. The question of the encephalitis neonatorum of Virchow, which is still under debate, cannot be entered upon here. Disseminated myeloencephalitis has already been described (pp. 320 and 340). Purulent encephalitis will be discussed in a special chapter.

All these forms are, therefore, to be passed over here, partly because they are treated of under other names elsewhere, and partly because in them the encephalitis is quite secondary to the principal pathological lesion. The important and distinct forms of encephalitis which are to be treated here are:

I. Acute Hæmorrhagic Encephalitis

The cause of this disease, which has been specially described by Strümpell, Lichtenstein, Fürbringer, and myself, is, as far as our knowledge goes, in all or in the majority of cases an *infection*. Cases of this kind were particularly observed during the *influenza* epidemic, this pathology being ascribed to them (although the symptoms corresponding to this were not by any means always present). Very likely the disease may just as readily develop during measles (Henoch), scarlet fever, pneumonia (Carré, E. Fränkel, Masetti), or from erysipelas. Encephalitis *after whooping-cough* has been repeatedly described, as by Neurath, Jacke, Hartmann, and Rhein; perhaps, one of Arnheim's cases also comes under this category. In a case diagnosed by myself the encephalitic process was proved by the post-mortem examination. The disease seems

also capable of developing after mumps (Putnam). Although on one occasion (Mendel) *post-diphtheritic* hemiplegia has been related to a hæmorrhage and is usually referred to embolism (Henoch, Slawyk, and others), it is undoubtedly the case that this paralysis is really caused by hæmorrhagic encephalitis. A few pathological reports (Krauss) and clinical observations (Oppenheim, Thiele, Muratow) point to this conclusion. According to Coester a relation between encephalitis and erythema nodosum must also be considered possible.

In other cases, in which no connection with any special infective disease could be proved, a relation to *cerebro-spinal meningitis*—to a certain extent an abortive form of this disease—has been assumed. The disease has been observed also in *ulcerative endocarditis*. I have noted it also in individuals suffering from *suppurative otitis*, and this has been confirmed by Voss and others.[1]

It is probable that the disease may also take its origin in the nose and accessory sinuses. Thus I have treated, in association with Jansen, a patient suffering from chronic suppuration of the sphenoidal sinus in whom a group of symptoms, which I could refer only to cortical encephalitis, developed after an operation.

This case is supported by an observation of Hirschl's, who confirmed the presence of a localised meningeal encephalitis in a case of putrid bronchitis. Observations of Fischl and Linsmeyer point to the appearance of encephalitis in septicæmia. Schmidt places the cases of encephalitis occurring in the course of septic-pyæmic diseases in a separate group. In a case described by Dana and Schlapp the disease was probably caused by malaria.

For the so-called meningo-encephalitis tuberculosa, *cf.* p. 784. But there seems also to occur a tuberculous form of hæmorrhagic encephalitis (Bombicci, Nonne).

Numerous observations point to the occurrence of a syphilitic encephalitis: Oppenheim, "Die syph. Erkr. d. Gehirns," ii. Aufl., Wien, 1903, Keller (*Inaug. Diss.*, Berlin, 1893), Rosenfeld (*Z. f. N.*, Bd. xxiv.), Barrett (*Amer. Journ. of Med. Sc.*, 1905), and others.

It is not improbable that in some of the cases we have to deal with an independent infective disease. Pfuhl discovered influenza bacilli in the central nervous system of influenza patients, and Nauwerk succeeded in isolating them in the foci of influenza-encephalitis. In a case of meningo-encephalitis E. Fränkel found accumulations of the diplococcus lanceolatus in the lymphatic sheaths of the small arteries (*cf.* Fig. 307); and Masetti has described a similar case. But it is also possible that secondary infection and the activity of the bacterial toxin may play a part in the production of encephalitis. Muratow even considers the latter—the para-infectious origin—as the regular one. Sträussler, R. Wagner, and others refer it to *anto-intoxication* following upon constipation. In a case of Ransohoff's it was due to dysentery or a similar affection. I have seen a serious form of the disease appear in a case of severe diarrhœa with vomiting. Raymond and Philippe allude to a senile form of acute encephalitis.

[1] The term encephalitis serosa, selected by Müller, Merkens, Lecené, Herzfeld, and others for the acute cerebral œdema appearing in the course of otitis, may lead to misconceptions, yet I consider it probable that serous meningitis (*q.v.*) may be associated with acute encephalitis, and I was recently able to settle this point with certainty by lumbar-puncture and brain-puncture in a case which took a favourable course.

The importance of injury as a cause of non-purulent encephalitis has not yet been made clear. In addition to the experimental investigations of Ziegler, Coën, Friedmann, and others, we have also clinical and anatomical observations made by Dinkler, Koeppen, Wiener, Rosenblatt, and Bethe, which place it beyond doubt that this encephalitis may result from an injury to the head—and, indeed, from a simple contusio capitis. The only question is: Is the traumatic form merely masked by the infective one? We must also think of the possibility that the trauma only offers a lesion which serves as a nidus for the agents of infection—a view which seems to follow from the experimental observations of Ehrenrooth.

Dexler (*M. f. P.*, xiii.) makes interesting statements on the occurrence of acute encephalitis in horses after glanders. I have myself observed one case of this kind—certainly not an entirely authenticated one—in which the disease seemed to have arisen through an infection from glanders in man; it was described by M. Bloch (*Ärztl. Sachverst.*, 1904). For the relations between Born's horse-disease and meningo-encephalitis, see Dexler (*Z. f. Tier.*, 1900) and Oppenheim-Ostertag (*Z. f. Inf.*, etc., Bd. ii.).

Symptomatology. The disease develops acutely and, as a rule indeed, in a fulminating manner, and attacks mostly *youthful* and previously healthy individuals. Children and young girls are perhaps the most frequently affected, and occasionally it is found in *anæmic* girls. It is, however, also found in later life, and even a senile form has been described—though that perhaps requires a special category.

Without any premonitory symptoms or after one or two days of headache, giddiness, depression, or irritability, the patient becomes dazed, and then *unconscious*, and the unconsciousness soon passes into *stupor*. Sometimes a *rigor* precedes the disturbance of consciousness. While this lasts the condition resembles an apoplexy, but the coma is rarely so deep—the light reflex of the pupil, the tendon reflexes, and, as a rule, the superficial reflexes are almost always present; the temperature, too, does not fall but is at once raised or rises as the disease proceeds; symptoms of paralysis, also, are usually absent or appear in a few cases at the commencement of the disease. During the attack *stiffness of the neck* may arise, and more rarely we have general or unilateral *convulsions*. Along with the stupor there may also be restlessness, confusion, and delirium. The respiration is usually quickened, and in some cases the Cheyne-Stokes type of breathing has been noticed. As a rule the pulse is abnormally rapid, but it may also be slowed—especially in the early stages. An enlargement of the spleen was found in only a few cases. The coma may become deeper and death ensue after the lapse of twenty-four hours to some days—with an ever-increasing temperature, specially just before death—without the patient having recovered consciousness, as I have myself seen in one case. A more protracted sub-acute course of the disease, however, also occurs, and even where there is a fatal end it has been proved that the disease may extend to twenty days, even to some months. In such a case *remissions* usually occur, and the fever is intermittent; in particular the brain sometimes clears a little, and *symptoms of paralysis* may now appear.

These vary according to the seat of the disease; we have chiefly *monoplegia, hemiplegia*, or *aphasia*—the last I have noted in quite a number of cases. I have also succeeded in describing a type of this disease which is characterised by the combination of general cerebral phenomena with

the focal symptoms of right brachio-facial monoplegia and motor aphasia. More rarely cortical epilepsy and hemianopsia, as in the observations made by Fürbringer, Mills, Spielmeyer, and in some cases treated by myself, were the focal symptoms. I have noted several times the presence of *optic neuritis* and more rarely choked disc. Sometimes other cranial nerves, *e.g.* the abducens, were affected.

More rarely, but still in a considerable number of cases, the pons, medulla oblongata, or even the cerebellum were affected. Leyden, Etter, and Eisenlohr had previously found inflammatory processes in these regions of the brain and described them as *acute bulbar-myelitis*, as *acute inferior polioencephalitis*—acute encephalitis or poliomyelitis. I have myself seen several cases of this kind—ending favourably for the most part. In describing disseminated myeloencephalitis (p. 320) it has already been pointed out that the localisation of the areas of inflammation in these regions frequently caused the group of symptoms associated with so-called acute ataxia.

In a case under my own observation *cerebellar symptoms* were prominent, together with *hemiataxia*, *nystagmus*, and *optic neuritis*. There also the disease ran a favourable course. Along with Nonne and Concetti I have been forced several times by the symptoms to diagnose a localisation of encephalitis in the cerebellum. This in some cases has been proved by autopsy—as in a recent case of Bethe's.

A case described by Lenhartz, and later on investigated by myself, had cerebellar ataxy after typhoid—this also had probably an encephalitic origin. We owe similar observations to Batten (*Br.*, 1905, and *Trans. Med. Soc. London*, xxviii.).

In certain cases it has been proved that the group of symptoms in pseudo-bulbar paralysis (*q.v.*) could be traced to multiple encephalitic areas (Schlesinger-Hori, Piperkoff, Pfaunkuch, *Z. f. N.*, xxxiii.).

Other cases have been observed, in which the symptomatology of the disease differed very little from that of softening and presented little that was characteristic (Strümpell, Raymond-Philippe).

Spielmeyer (*C. f. N.*, 1904) lays stress on the fact that at times epilepsy, particularly cortical epilepsy, may dominate the clinical picture. To Mills (*R. of N.*, 1907) we are indebted for an interesting observation of the same kind. Cases also occur in which psychical disturbances are very prominent (Preobrajensky, ref. *N. C.*, 1906). Finally, a few cases have been described by Hegler-Hebber (*A. f. kl. Med.*, Bd. xxxii.), and Laache (*Nord. Mag. f. Laeger*, 1905), in which the symptoms closely resemble those of tubercular meningitis.

An *acute inflammatory condition* in the brain, pre-eminently *hæmorrhagic* in character, forms the basis of the *morbid anatomy*. It is usually in circumscribed areas, and frequently limited to symmetrically situated regions of the brain. Thus, it is frequently found in the centrum semiovale, in the cerebral cortex, and specially often in the central ganglia. As a rule, then, it is the cerebrum which is affected. Without doubt, however, the same process may take place in the brain substance, and particularly in the central grey matter of the aqueduct of Sylvius (see below), in the pons, and in the oblongata. Up till the present time it has been least frequently found in the cerebellum. The tissue affected appears even to the naked eye to be markedly hyperæmic, with marked puncta cruenta (see Fig. 304), generally to be swollen and more moist than normal. Besides the numerous small hæmorrhages there occur at times more

extensive ones ; indeed, it is by no means improbable that here and there amongst cases—especially among young individuals—diagnosed as cerebral hæmorrhage, there have been cases of unrecognised hæmorrhagic encephalitis (Dexler). On the other hand, Schmidt would rather give to some of the cases of so-called hæmorrhagic encephalitis the term cerebral hæmorrhage. Only in a few cases were changes visible to the naked eye awanting. *Microscopically* the vessels—the small arteries and capillaries—are first of all affected : they are dilated and engorged with blood, which distends the vessel sheath and, after it has ruptured, passes into the surrounding tissue : besides this an infiltration of white blood

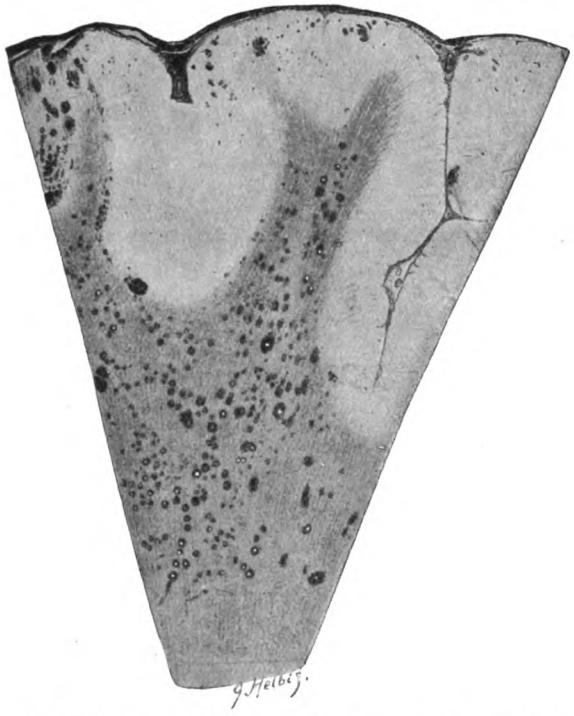

FIG. 304.—Petechial encephalitis (microscopic section).

corpuscles and, in more prolonged cases, granular cells and proliferated glia-cells are found.

Recently I had the opportunity of observing this in the living subject : in a case in which I had given a diagnosis of encephalitis of the left temporal lobe (and meningitis serosa), a puncture carried out by Borchardt yielded a *dark, oily fluid* tinged with blood, which microscopically showed abundance of lymphocytes and *granular cells*. It originated in the white matter of the left temporal lobe.

Signs of irritation and disintegration are to be found in the nervous elements. When one considers that a complete " restitutio ad integrum " is possible, even where the signs point for instance to an extensive interruption of the conduction in the pons, one must assume that the pathological changes are slight and that they do not pass beyond the first stage in those cases which have a tendency towards healing. That the process

may, however, result in cicatrisation or rather sclerosis, is proved by Figs. 305 and 306, which refer to a clinically recovered case observed by ourselves.

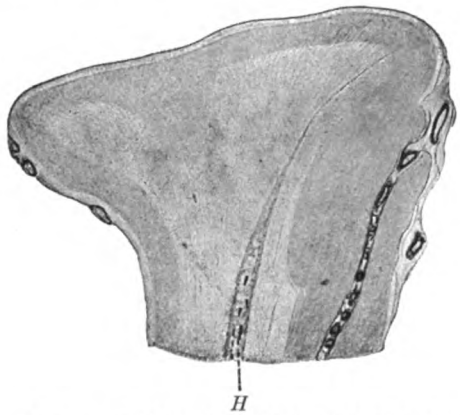

FIG. 305.—Acute hæmorrhagic encephalitis of the left frontal lobe in the stage of cicatrisation. *H*, Focus of hæmorrhage. (Carmin-alum hæmatoxylin stain.)

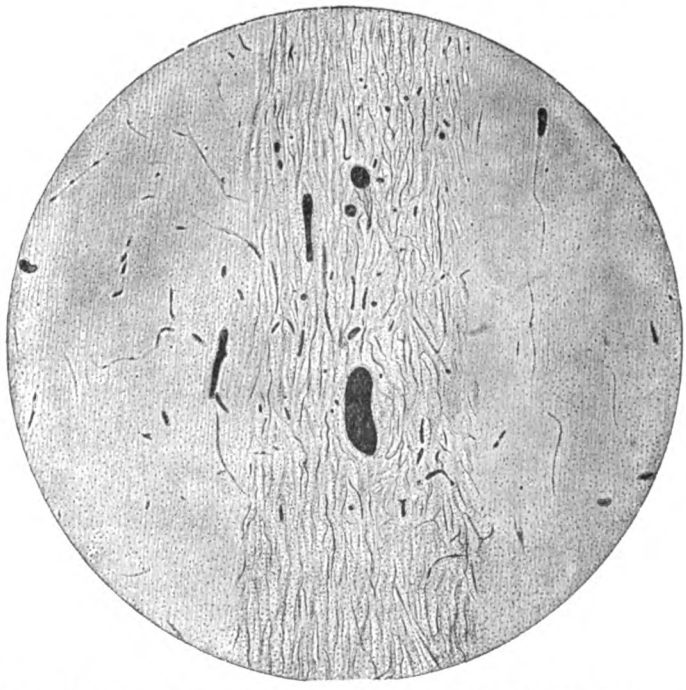

FIG. 306.—Part of cicatricial focus from preceding figure under stronger lens. (Carmin-alum hæmatoxylin stain.)

As a matter of fact localised diseases do occur, which anatomically cannot be distinguished from softening, except that they cannot be referred to any blocking of the vessel-lumen. I have myself observed this condition, and it has also been described by Köppen, Piperkoff, and others. An

observation, made by Henneberg, in which cerebral disease was combined wth myelitis, points to the occurrence of an encephalomalacic form of encephalitis.

Hayem, Friedmann, Raymond and Philippe, M. B. Schmidt, and others have laid special stress on the appearance of large epitheloid cells in encephalitic areas (hypoplastic form of encephalitis). The question whether these have their origin in the proliferation of the fixed tissue-cells or are the descendants of leucocytes is not yet decided. A case described by Spielmeyer appears to belong to this type. It can scarcely be longer doubted that the so-called infantile paralysis (*cf.* next chapter) is in many cases the effect of an acute encephalitis, localised in the motor areas of the brain (Strümpell, Marie, and Batten take this view). Ganghofner, Fischl, Raymond,[1] and Weyl lay stress on the occurrence of acute encephalitis in the early years of childhood. It is unquestionable, however, that there are forms of it which vary both as to its nature and distribution. Thus in some cases—as in one described by Fischl—there is a diffuse process, which extends over both hemispheres. In

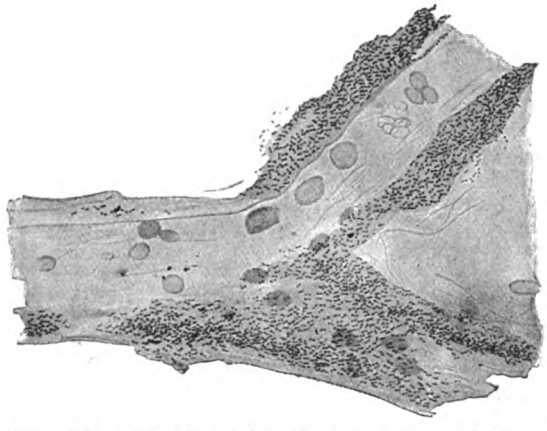

FIG. 307.—(After Nonne-Luce, or E. Fraenkel.) Meningo-encephalitis from diplococcus lanceolatus (case of Eugen Fraenkel's, by whom the drawings were made). The pneumonia-carriers, which line the vessel like a sheath, occupy the dilated extra-vascular lymph-space. (Red and white blood corpuscles in the lumen of the vessel.)

another case he was able to refer the cerebral symptoms to an invasion of the brain by streptococci in large numbers: his experience indeed has led him to look upon septic infection or intoxication as the main cause of the disease. Raymond thinks that diffuse encephalitis may issue in diffuse cerebral sclerosis (p. 344). Finally, even disseminated myelo-encephalitis is not infrequently observed in the early years of childhood.

Sinus thrombosis may also be combined with encephalitis (observations by Siemerling and Oppenheim. Bückler, Nauwerck, Laache, etc). Its combination with thrombophlebitis is described by Muratow. A general hæmorrhagic diathesis can often be traced (Sträussler). In the case of "encephalo-myelo-meningitis diffusa hæmorrhagica," described by Bartel, the author himself considers a syphilitic origin as probable. Sometimes a marked effusion into the ventricles was present—a combination of

[1] See also his latest publication in *Arch. de méd. des enf.*, x.

encephalitis with meningitis serosa (R. Müller, Leyden, Nonne, Oppenheim), meningeal hyperæmia, etc. Eichhorst, in particular, has shown the connection with meningitis, and refers the encephalitic process to a primary hæmorrhagic inflammation of the pia. In a case investigated by Dana and Schlapp there were multiple areas of inflammation in the brain and spinal cord. A combination of encephalitis with acute anterior poliomyelitis has also been under observation (Lamy, Redlich, Beyer, Oppenheim, Marie, Rossi [1]).

We can do no more than allude here to the exceedingly rare *blastomycosis cerebri*—those modifications of the cerebral tissue caused by accumulations of yeast-cells—observed by Hansemann and Benda (*D. m. W.*, 1907) and also Türk (*A. f. kl. M.*, Bd. xc.).

I cannot agree with the hypothesis that direct transitions occur between encephalitis, meningitis, and abscess (Raymond-Cestan, *Gaz. des hôp.*, 1904). On the other hand my own experience agrees with the view that a cerebral abscess (*q. v.*) is frequently accompanied by areas of non-purulent hæmorrhagic encephalitis.

As regards other organs, we find, associated with encephalitis, splenic enlargement, nephritis, parenchymatous degeneration of the myocardium, and such like conditions.

A *diagnosis* of acute hæmorrhagic encephalitis should be made only with the greatest reserve. It is specially difficult and—as Nonne has pointed out—in certain cases quite impossible without gross anatomical changes (*cf.* p. 868) to distinguish it from acute serous meningitis, from sinus-thrombosis, and from the group of symptoms (pseudomeningitis) which appears after infective illnesses and is so like meningitis. One must always keep in mind the fact that the cerebral symptoms, which appear as a result of acute infective illnesses such as influenza, typhoid, pneumonia, or whooping-cough, may have either a purely toxic origin or be caused by hæmorrhages and embolic-thrombotic processes. Encephalitic processes are not always so sharply defined from these processes as to make a certain diagnosis possible. On the whole I have the impression that the diagnosis of encephalitis is often too rapidly arrived at. The differentiating characteristics will be found in the corresponding chapters. Above all a distinguishing feature of encephalitis as opposed to the condition of intoxication is that in it focal symptoms usually develop early and are very marked.

In the rare cases in which the disease runs a sub-acute or even chronic course, it may easily be mistaken for a cerebral tumour, especially when it is associated with a serous meningitis. But if the fact of its having had an *acute and febrile onset* can be verified, one should adhere to the encephalitic diagnosis.

A case of Rosenfeld's is exceptional, because in it the encephalitic process was limited to the optic nerve or optic tract.

It must, however, not be forgotten that in encephalitis all the symptoms are not based on demonstrable pathological changes, but that the infection or intoxication produces a general injury to the nervous system without any structural changes (Oppenheim, Nonne, Schmidt).

The *prognosis* of hæmorrhagic encephalitis is grave. In cases with an abrupt and severe commencement, pronounced disturbance of consciousness, and high fever, the issue is usually a fatal one. On the other hand numerous cases observed by myself, with which others described by Fürbringer, Fraenkel, Feryhan, Thiele, Nonne, Stegmann, Bregmann,

[1] *Nouv. Icon.*, xx.

and others agree, prove that recovery is not infrequent. In a case of acute encephalitis, confirmed by post-mortem investigation, a cure was effected, and I found a scar as the outcome of a process which had run its course (Figs. 305 and 306). Subsequently Koeppen and Friedmann have published similar observations, and the latter has also noted the formation of cysts as a consequence of encephalitis. Again, I have recently had the opportunity of observing, during surgical operations, cysts without walls in the tissue both of the cerebellum and the cerebrum, which possibly had an encephalitic origin (in two cases post-traumatic). Muratow remarks that the prognosis is rendered more serious by the presence of larger hæmorrhages. Convalescence may extend over weeks, months, or even years. Recovery, with permanent defect, also occurs. It is, in fact, the rule if we include here the Strümpell form of cerebral infantile paralysis. Relapses seem also to occur (as shown by my own and Dinkler's and Wiener's observations).

In the prognosis we must further on no account leave out of sight the fact that there are forms of encephalitis which bear a close relation to multiple sclerosis. This is true chiefly of disseminated myelo-encephalitis (see p. 320) and of encephalitis of the pons and medulla oblongata. Unfortunately we lack sufficiently sure data to be able to decide in the acute stage or immediately after that has subsided, whether the recovery will be a lasting and complete one or whether the disease will pass over into multiple sclerosis. Compare a case observed by Maas and myself, and afterwards described by him (*M. f. P.*, xviii.).

As to *treatment*, little definite can be said as yet. It is in any case desirable to afford the patient a full measure of rest and care. He should lie in a room free from noise and intense light, and all mental excitement should be avoided. The therapeutic measures to be adopted at the beginning are cold compresses or an ice-bag to the head and blood-letting by venesection or leeches. The latter is contra-indicated only in the case of very anæmic persons. In a very serious case under my own care, improvement began with the use of warm baths. As to the use of drugs we have no sufficient experience, but it would be quite proper to give salicylates, quinine, and antipyrin. In some of our most successful cases we have used large doses of *calomel*, besides blood-letting, until stomatitis was induced. In this disease it seems to us quite justifiable to try unguent. argent. colloid (Crédé) . Hot footbaths may be ordered in very protracted cases.

In one of our cases, to which I have already alluded above, a favourable change in the patient's condition immediately followed *puncture* of the focus, when about 5 to 10 c.cm. of cloudy fluid were withdrawn. I do not, however, by any means wish to imply that surgical treatment should always be adopted for this disease, which is spontaneously curable; I desire only to recommend puncture for the serious cases in which there is an " indicatio vitalis."

The treatment of the resulting conditions has already been fully discussed.

II. Poliencephalitis Acuta Hæmorrhagica Superior

For literature see Oppenheim-Cassirer, *l. c.*, and Uhtoff, Graefe-Saemisch, " Handbuch " 2nd edition, Bd. xi.

Wernicke, assisted by an observation already made by Gayet, first recognised the independence of this condition. Thomsen, Boedeker,

Eisenlohr, Jacobaeus, Goldscheider, Bonhoeffer, Gudden, Raimann, Wijnhoff-Scheffer, Brissaud-Brécy, Hunt, and others have added further accounts.

In cases of this kind the acute hæmorrhagic encephalitis is limited to *the region of the central grey matter* around *the third ventricle* and *aqueduct of Sylvius*, or extends down into the fourth ventricle and even into the grey matter of the spinal cord. The inflammatory changes are less prominent in this disease, while new-vessel formation and small hæmorrhagic foci are the most marked features. Spielmeyer, who points out these facts, places the process in the group of diathetic hæmorrhages—a view which has been discussed fully by Fränkel, Redlich, and others. This form is taken here separately because, both from its ætiology and symptomatology, it takes a distinct position, but the differences between it and those previously described are by no means marked. *Chronic alcoholism* is the most important cause. Most of those who suffer from it have been tipplers; in one case the illness was connected with poisoning by sulphuric acid. Perhaps other poisons (meat, fish, or sausage poisons and such like) may also produce it. In one case I saw all the symptoms in a patient, who had been treated for a prolonged time with lysol, but I was unable to prove the ætiological connection with any certainty. Further investigation (Gayet, Uhthoff-Oppenheim, and others) teaches that the same form of encephalitis may arise also as a *result of infective diseases*, especially *influenza*, and is distinguished, therefore, from the one first described by the difference in localisation.

Let us next consider the clinical symptoms in the typical form of the disease, namely, that first described by Wernicke, Thomsen, and others.

The disease begins acutely and usually runs an acute course, ending, as a rule, fatally within 8 to 14 days, although recovery has been seen in a few cases. After a considerable time, in which the signs of chronic alcoholism have been evident, or perhaps after only a few days of headache, giddiness, and vomiting, and even without any such premonitions, a disturbance of consciousness sets in in the form of *delirium* or simple *somnolence* with restlessness. More rarely apathy and lethargy are seen. Stiffness of the neck occurs in a few cases. At the same time *symptoms* of *paralysis* may develop in *the muscles of the eye*—more frequently associated paralysis—which may increase to an almost total *opthalmoplegia*. It is not uncommon, however, that certain muscles, such as the levator palpebræ superioris, and particularly the sphincter iridis, should escape. Optic neuritis may also be present, and nystagmus has been several times noted. A certain locomotor disturbance, which recalls *cerebellar* ataxia, is a very usual sign of this disease. Further, weakness, tremor, and ataxia in the limbs may be noticed. The articulation is frequently impaired. In a few cases there was partial facial paralysis and in others hemiplegia.

The deep reflexes are normal or exaggerated: they are absent only in exceptional cases. The temperature is almost always normal or lower than normal. Fever is unusual. The pulse is usually accelerated and, particularly towards the end of life, small and rapid. Tachypnœa and other respiratory disturbances now also develop.

Bing (*B. k. W.*, 1906) found, in one of our cases, considerable diminution of the blood-pressure.

The disease pursues an ultra-acute or acute course, and usually ends

in death in a period of from 8 to 14 days, but a more protracted course with ultimate recovery may occur.

In one serious case of this kind I saw all the paralytic symptoms disappear, but a severe loss of memory persisted. Other writers have pointed out how frequently a psychic defect continues after recovery from paralytic symptoms (Bonhoeffer, Spielmeyer).

In cases investigated so far a hæmorrhagic inflammatory process of the kind depicted above has been found, limited to the central grey matter of the third ventricle, of the aqueduct of Sylvius (Figs. 308 and 309), but occasionally extending to the fourth ventricle. Sometimes, instead of the hæmorrhagic inflammation, a degeneration of the corresponding nuclei of origin of the nerves could be demonstrated.

It is, however, to be noted that the affection does not by any means

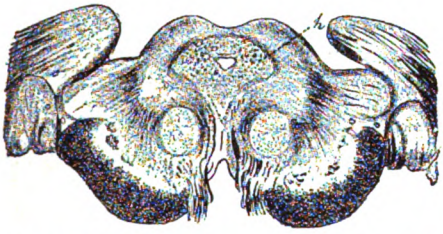

FIG. 308.—Acute hæmorrhagic polio-encephalitis superior. *h*, Site of the encephalitic process. Spotted appearance caused by the numerous foci. Carmin section in Oppenheim's collection.

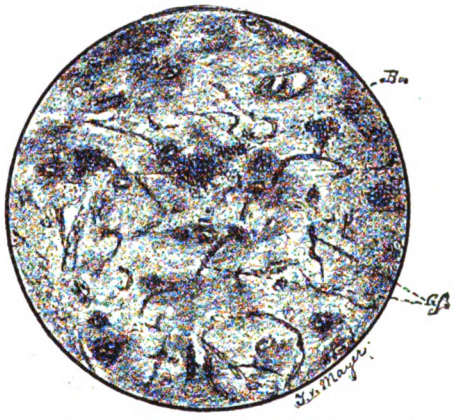

FIG. 309.—Acute polio-encephalitis superior. From a high-power enlargement of a carmin section in Oppenheim's collection.

keep strictly to the limits of the grey matter, but on the contrary passes over to involve the white matter—showing in this a further analogy to acute poliomyelitis, with which also it may be combined. On the other hand, it is an established fact that this poliencephalitis develops frequently as a sequel to polyneuritis, just as by way of variety, a neuritis of isolated cranial nerves may be combined with the former.

Raimann goes so far as almost always to attribute the paralysis of the eye muscles which occurs in alcoholism to poliencephalitis. He also makes the statement, which seems to us surprising, that the intrinsic muscles of the eye are frequently affected in alcoholics.

In a further series of cases, which certainly come under this category, the disease followed *influenza* or some other *infective disease*; thus an investigation made by Luce shows that poliencephalitis hæmorrhagica superior may develop along with tubercular basal meningitis. I have once seen this condition follow perityphlitis. In other cases, again, it was impossible to assign any particular cause.

The development here is not always a rapid one, but may extend over weeks and sometimes even over a longer period. Stupor and any

indication of general disease of the brain may be absent. In the majority of these cases—so far as it was possible to draw any conclusions from the clinical signs and the scanty information supplied by a post-mortem examination (Kaiser, Saenger)—the process extended and involved the origin of the upper cranial nerves and also the lower ones, namely, those which lie at the floor of the fourth ventricle, and there developed *poliencephalitis superior* and *inferior*, and in some cases even *poliencephalomyelitis* (poliencephalitis superior, inferior, and poliomyelitis anterior or myelitis). Thus Uhthoff and I observed a case in which difficulties in swallowing and speaking were associated with paralysis of the muscles of the eyes—which nevertheless resulted in recovery. Similar observations have been made by Etter, Goldflam, Kollarits, Muratow, Zappert, and others, and very frequently by myself.

In the symptomatology of this affection the focal *symptoms* are the most important. They point to a morbid process extending in rapid succession from the nuclei of the nerves of the mesencephalon to those of the pons and medulla oblongata. There are thus the manifestations of *ophthalmoplegia* and *bulbar paralysis* which appear here in varied groupings and combinations. In general the course is a descending one, but occasionally it is reversed. The *general symptoms* correspond to those described at the beginning of this chapter; they may, however, be very slightly developed or even be altogether absent. As a rule the attack is accompanied by a rise of temperature. The course is either acute or sub-acute; sometimes it goes by fits and starts. As the anatomical process is, however, by no means always limited to the grey matter, but sometimes spreads diffusely into the pons and medulla oblongata, so the clinical manifestations correspond frequently to a diffuse disease of these regions, and we have the same clinical picture which we have already described in another place as appearing in localised diseases of the pons and of the medulla oblongata. The various forms of alternate hemiplegia are specially apt to be produced by these affections. Ataxia, also, in its different types, may constitute a main element in the symptomatology.

In so-called *poliencephalomyelitis* we are dealing with a paralysis extending in a more or less symmetrical manner over a portion of the cranial and spinal motor nerves. An affection of the cranial nerves produces the clinical picture that belongs to ophthalmoplegia and glosso-pharyngo-labial paralysis, and the spinal affection causes either diffuse or circumscribed, chiefly atrophic, spinal paralysis. Sometimes one and sometimes another component develops more strongly amongst the symptoms. An electrical test gives as a rule only a quantitative decrease of excitability or partial reaction of degeneration. Since, however, the anatomical process is not limited to the grey matter, the clinical manifestations (loss of sensation, etc.) may also offer a proof of the non-systemic character of the lesion.

In the majority of the cases of this kind recorded up till now (Rosenthal, Seeligmüller, Guinon, Parmentier, Kalischer, Schaffer, Taylor, Roth, Klippel, Goldstein, and others) the disease pursued a subacute or chronic course. But a few cases have also been described in which the course was acute, and the similarity with Landry's paralysis was striking (Green-Wilson, Sherman-Spiller, E. W. Taylor). Recovery took place occasionally, where the course was subacute. Cases of a more chronic kind will be considered in another place. Compare the chapter on opthalmoplegia.

The *diagnosis* of poliencephalitis and poliencephalomyelitis acuta should be made only with great reserve, seeing that there are diseases of very similar aspect in which actual gross changes cannot be found. Probably these arise mostly from toxic-infectious conditions without any anatomical basis. In especial a large number of the cases of poisoning from fish, meat, or sausages, which show paralytic conditions as a result, and which correspond more or less perfectly in this respect to poliencephalitis superior, inferior acuta, and poliencephalomyelitis acuta, should be included among them. Cases of this kind have been described by Cohn, Leber, Alexander, Guttmann, Lauk, and others (see the exact bibliographical references by Uhthoff, *l.c.*), and more recently by Preobrajensky, as ptomaine-paralysis (paralysis from septic poisoning), without confirmation by post-mortem examination. Fajersztajn has also published interesting observations of the same kind. Auto-intoxication from the intestine has also been given as a cause (Raimann, Sträussler, and others).

Cerebral symptoms may be produced in childhood by the bacillus coli or its toxin—and these may very closely resemble the symptoms of acute encephalitis (Seitz). A local invasion of micro-organisms instead of the expected inflammatory changes (Eisenlohr, Seitz) has occasionally been observed.

The question of the system relationship of polioencephalitis superior acuta to sleeping-sickness can only be mentioned here.

The two chief forms of encephalitis which have been depicted, namely those of Wernicke and of Strümpell-Leichtenstern, cannot be sharply divided from one another. There are transition forms and mixed forms, and both may be united in one case.

Cerebral Infantile Paralysis

Hemiplegia and Spastic Infantile Diplegia, Infantile Cerebral Paralysis

For bibliography see Freud, "Die infantile Zerebrallähmung," Nothnagel's "Spezielle Path. und Therapie," Bd. ix. 3; and Collier, "Cerebral Diplegia," *Br.*, 1899. The works not quoted by these authorities are nearly all mentioned in the text of this chapter.

This form of cerebral paralysis—congenital or acquired in early childhood—is not characterised as a special disease by any definite pathological changes, but by its clinical features and its course. It commences in rare cases during fœtal life, more frequently during labour, but usually its symptoms make their first appearance during *the first years of life*, whether the disease, as in the majority of cases, is acquired in after life, or whether the symptoms of a congenital condition become evident shortly after birth. Therefore, we can with Sachs distinguish the paralyses occurring antenatally, during labour, from those subsequently acquired; but this differentiation is by no means always practicable.

As regards the ætiology, our knowledge is still defective. *Heredity* does not seem to be of any great importance, but I have certainly seen one instance in which a mother and a daughter suffered from the disease; on the other hand there are certainly hereditary and family diseases of the nervous system, which are nearly related to this affection. Amongst the *noxæ* which lay the foundation of this disease in fœtal life, the most important is *injury* (injuries to the gravid uterus). Emotional

excitement of the mother during pregnancy has also been blamed. But *syphilis* in the parents, or *hereditary syphilis*, seems a very common cause of the disease (Erlenmeyer, Osler, W. Koenig, Vizioli, Massol).

The injuries that occur during birth are, however, of much greater importance. *Premature birth*, *difficult delivery* with prolonged delay of the head above the brim, difficult delivery of the head after a breech-presentation, extra-uterine pregnancy (Grósz), the birth of twins (Dösseker saw the affection in a triplet), asphyxia neonatorum, are all conditions which frequently give rise to this condition. In a case under my own care it could be traced to hæmorrhage from the umbilical cord. The use of the forceps has also been blamed, but the actual use of the instrument is less responsible for causing the disease than the difficulties which render its use necessary. These injuries act especially

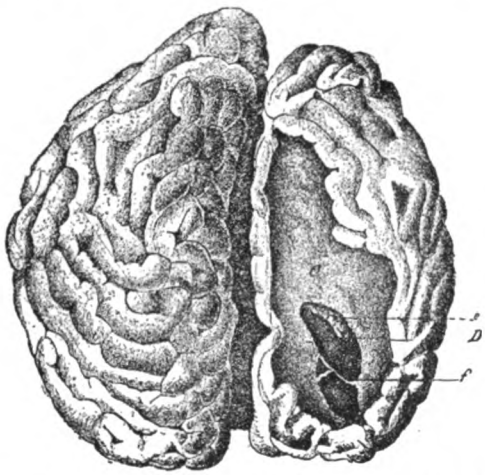

FIG. 310.—(After Ferraro, reproduced by Starr.) Defect and atrophy of the right hemisphere in a case of porencephalitis. At *D* the cavity passes into the lateral ventricle where the choroid plexus *e* is visible.

by producing *meningeal hæmorrhages*, which affect the meninges over the motor zone and cause lesions in the cortex (Sarah M'Nutt, Cushing). The hæmorrhage arises usually from rupture of the veins before their entrance into the sinus (Virchow).

Overlapping of the parietal bones may, according to Kundrat, cause compression of the longitudinal sinus even in normal deliveries. In this way the entrance of the venous blood into the sinus is rendered difficult, and occasionally even the veins which flow into it may be ruptured.

The most important etiological factors during *extra-uterine life* are *infective diseases*. In a relatively large number of cases the affection develops during the course of acute infective disease or as a consequence of it. *Measles* and *scarlet fever* with nephritis or endocarditis have frequently preceded it, but whooping-cough, small-pox, pneumonia, and other conditions, may give rise to it. It is said that even vaccination has occasionally produced it. It is probable that micro-organisms are conveyed to the brain by the blood-vessels. It is difficult to decide what part is played by *embolic processes*. Emboli (in endocarditis)

have several times been found in the branches of the artery of the Sylvian fissure.

A number of observations show that an encephalitic process of an infective nature is probably the cause of cerebral infantile paralysis—indeed, we have here an acute form of non-purulent encephalitis, which affects chiefly the motor areas of the brain (cortex and medulla, more rarely the central ganglia), (compare last chapter). This acute encephalitis of the motor area of the brain has been shown by Strümpell—and previously by Benedikt and Vizioli—to have an analogy with acute anterior poliomyelitis, and to form the typical change in a well-marked variety—well marked even clinically—of cerebral infantile paralysis. Marie and Batten have also laid stress upon this. Raymond claims to have demonstrated the presence of a pathogenic bacillus in the diseased

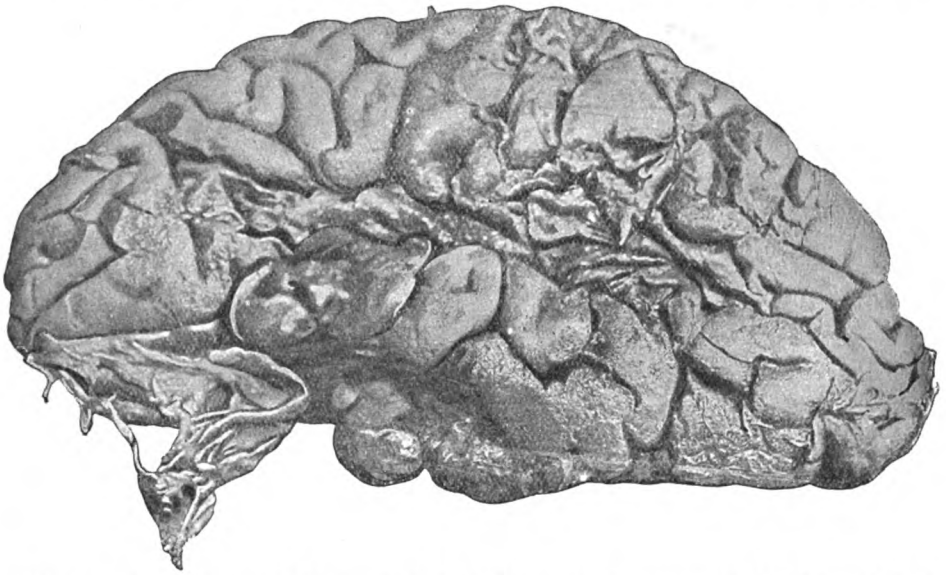

FIG. 311.—Extensive area of destruction of the left hemisphere in porencephalitis. (Oppenheim.)

brain area in a case of this kind. Gowers, on the other hand, takes it for granted that a thrombosis of the arteries, or of the veins, or of the sinus, forms the starting-point of the disease, and other writers believe it to be of hæmorrhagic origin.

Cranial injuries may even in extra-uterine life give rise to its development. At any rate, after injury to a child's skull, a group of symptoms may develop, which cannot in any way be distinguished from the ordinary forms of spastic infantile hemiplegia. Corresponding pathological conditions, *e.g.* porencephaly of traumatic origin, have been described (Kahlden and others). Fright has also been alleged to be a cause. Regarding the question of the ætiology in more recent treatises, see Fränkel (*Z. f. orth. Chir.*, xv.).

Pathological Anatomy.—All cases cannot be attributed to a single pathological cause. The great majority of the conditions to be considered are related to a state already past; in some a localised disease of the

cerebrum was found; in others a centre of softening; one or more *cysts*, mostly of hæmorrhagic origin, cicatricial shrinking and *induration*, or a *defective formation* of the cortex (Figs. 310 and 311), etc. The latter condition, known as *porencephaly* (Heschl, Kundrat), occurs both in a unilateral and bilateral form (and is frequently symmetrically bilateral), most often in the central area or in the region supplied by the artery of the Sylvian fissure. The right side (Kellner) is most often affected; here irregular, funnel-shaped depressions are found on the surface of the brain, which dip in usually as far as the ventricle. The adjacent convolutions may converge towards the defect. The affection is usually ante-natal, but may be acquired after birth.

To distinguish between these two forms a number of data have been suggested (Kundrat, Kahlden, Bourneville-Schwarz): The funnel-shaped formation of the "porus," the point of

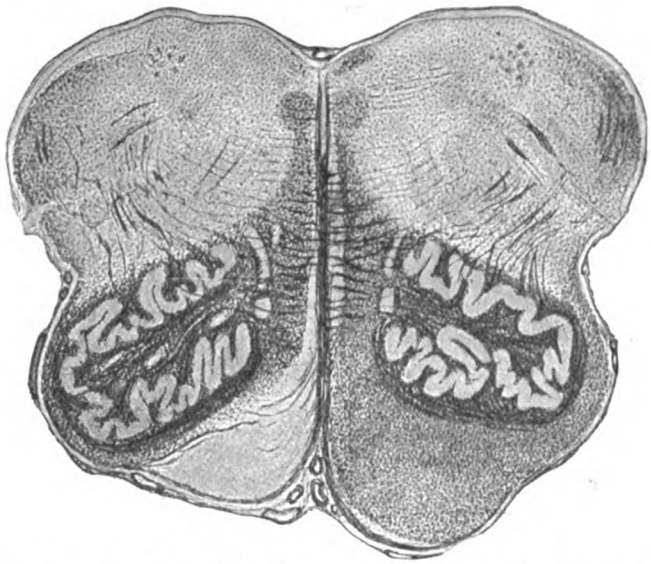

FIG. 312.—Atrophy and degeneration of the left pyramid in cerebral paralysis of childhood.

which is turned towards the ventricle; the radial arrangement of the convolutions in the direction of the base; combination with other malformations (microgyria, absence of corpus callosum), etc.—all point to an intra-uterine genesis. These true porencephalies are distinguished from the false ones, which, though also congenital, may just as well be acquired in extra-uterine life. Many writers (Beyer and others) doubt, however, the value of these distinguishing features. Recently Richter, Gangitano Zipfel, Lapinsky, Schroeder, Liepmann, Schupfer, Obersteiner, Schütte, Kotschetkowa, Bischoff, Wiglesworth, Weill and Gallavardin, Zahn, Kluge, Shirres, Kellner (*M. f. P.*, xii), Zingerle (*A. f. P.*, xxxvi.), Dannenberger (*Klin. f. psych. und nerv. Krank.*, Bd. i.), Baird, Messing (*Obersteiner*, xi.), and others have described such cases. Richter (*N. C.*, 1898) has advanced a new theory of the production of porencephaly, which—if it holds good at all—does so only in the minority of cases. Obersteiner ("Arbeiten," etc., viii.), in his case, ascribes to hydrocephalus an important part in the formation of porencephaly.

Kundrat has ascribed the disease to necrosis arising from occlusion of a vessel, but that is certainly not its only mode of origin. Probably encephalitic and meningo-encephalitic processes may lead to it, particularly when they occur in the fœtus. It has been ascribed by Heubner

and others to embolic processes. Injuries at birth are causes of considerable importance.

Malformations of a peculiar kind sometimes appear in the cerebral cortex, *e.g.* slight folding of the cortical grey matter after the manner of the formation of the convolutions in the vermis of the cerebellum (*microgyria*) (Fig. 313).

As regards the finer changes in this arrest of development, compare Fig. 314 and Fig. 315 (also Peritz: "Pseudobulbär- und Bulbärparalysen des Kindesalters," Berlin, 1902, S. Karger).

The anomalies of development described by Mierzejewski, Heschl, Chiari, Binswanger, Otto, Oppenheim (*N. C.*, 1895), Heubner, Obersteiner (*N. C.*, 1902), Clark-Prout (*Journ. of Amer. Med.*, 1902), May (*Brit. Med. Journ.*, 1905), Liebscher (*Z. f. Heilkunde*, Bd. xxvii.), and others as *microgyria* have been interpreted in various ways. My view is that the main factor is a process taking place on the surface of the cortex (meningoencephalitis, meningeal hæmorrhage), which causes

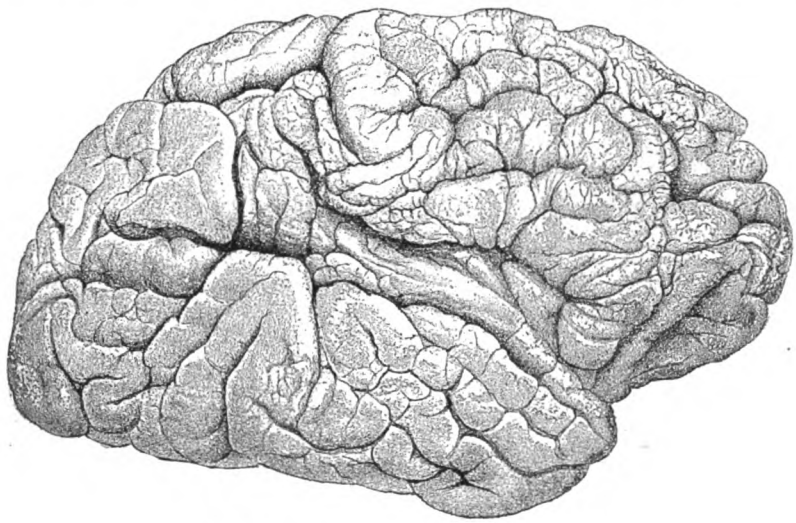

FIG. 313.—Microgyria. (After Otto.)

it to become shrunken, folded, and wrinkled. This view has been accepted by many of the most recent observers. How much the nervous elements of the cortex, especially the ganglion cells, may suffer under this anomaly of development is shown by Figs. 315*b*, 316, made from a preparation of my own. Mierzejewski and Kotschetkowa (*A. f. P.*, xxxiv.) and Marinesco, whom my description has escaped, have reported similar conditions. Koeppen, Kalischer, Bresler, Liebscher, Lapinsky, Probst, Schütz, Marinesco, Obersteiner, Gianelli (*Riv. sper. di Fren.*, 1901), and particularly Kotschetkowa, have recently contributed to the study of this subject. The last-named writer distinguishes between a primary microgyria, which represents a true arrest of development, and a secondary one of encephalitic origin. Obersteiner also draws a distinction between true and false microgyria. The observations and investigations of Oeconomakis (*A. f. P.*, Bd. xxxix., and "Die Mikrogyrie," Athens, 1906) also deserve attention.

For the relations of microcephaly to cerebral infantile paralysis, see *e.g.* Ibrahim (*Jahrb. f. Kind. N. F.*, x.).

Anomalies of the membranes of the brain, *e.g.* thickening and formation of cysts within the meninges, are often also present.

The process rarely takes the form of a circumscribed localised disease. The whole hemisphere, or a great part of it, is usually involved in the change, becoming decreased in size (in toto) as the result of a universal atrophy

(Fig. 310). This again is due to a chronic inflammatory process, viz., sclerosis. There are cases in which there is no localised disease, but merely this general induration—the so-called *lobar sclerosis* (Bourneville, Richardière, Cotard, Jendrassik-Marie, Bischoff, Spiller, Bouchaud, Felsch,[1] Köppen [2]).

Hypertrophic sclerosis, in which nodules arise in the brain substance, is a variety of this condition. *Hydrocephalus* occurs sometimes in combination with the changes already mentioned—very rarely alone. We also hear sometimes of simple *agenesis corticalis*, i.e. defective development of the nervous elements which reveals itself chiefly microscopically (Sachs, Massalongo, Collier [3]).

With regard to the *localisation* of this pathological process, the cortex of the *motor zone* is as a rule involved; it is but seldom the only part affected, as the disease is usually more widely diffused both in surface extent and in depth. Occasionally the sclerosis is localised in another region, *e.g.* in the central ganglia, and in that case the motor tract becomes destroyed.

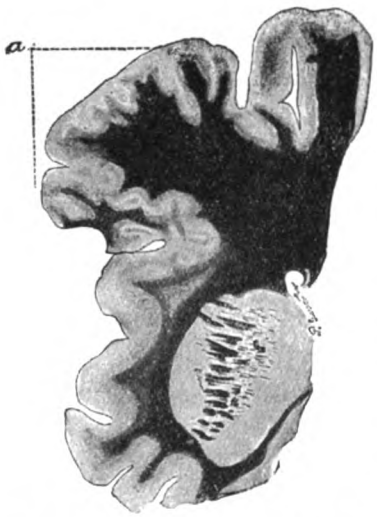

FIG. 314.—Frontal section through a hemisphere in microgyria. *a*. a site of the microgyria. Condition of the grey matter of the brain and the subcortical fibres in this anomaly of development. (From section stained with Pal's method. Oppenheim.)

In a case described by Bischoff the optic thalamus was affected, in another the cerebral peduncle. Dejerine and Thomas [4] found the lesion in the internal capsule in a case of this nature.

A case observed by Marie-Guillain (*Nouv. Icon.*, xvii.), in which the sclerosis was situated in the red nucleus, must also be included here on account of its symptomatology.

Porencephaly has also been found outside the motor region, *e.g.* in the occipital lobe, by Moeli and Richardière, and by Monakow and Anton (*M. f. P.*, xix.) in the cerebellum. The cases of Obersteiner, Messing, and Turnbull are also interesting in this connection (*Br.*, 1904). Naturally the clinical symptoms vary in character according to the site of the disease. In some cases congenital blindness and deafness and cerebral inco-ordination may be attributed to such changes.

Our information concerning the *initial lesion* is much less complete. It may be considered proved that hæmorrhage into the brain-substance and the membranes (M'Nutt), embolism and thrombosis with secondary encephalomalacia, encephalitis, and meningo-encephalitis may be the cause. If these disturbances develop in infancy or childhood, and if they affect the motor area (or nerve tracts), they are recognised by their symptoms—the phenomena of spastic infantile paralysis. Similar processes may occur in fœtal life and may lead to arrest of development, but it is probable that primary atrophy and arrest in the development of the cortical nervous elements is here an important factor (Mya and Levi, Sachs, Collier).

At a later stage it is not always possible to recognise the nature of the original disturbance, as similar consequences, such as induration and

[1] *A. F. P.*, xxxvi. [2] *A. F. P.*, Bd., xl. [3] *Br.*, 1899. [4] *R. n.*, 1900.

atrophy of the hemispheres or a great part of them, result from all the above-mentioned changes. Finally, we should specially notice that all injuries which reach the brain of a child have a most injurious effect upon the motor tracts and the pyramidal fibrils, and retard their development, especially in regard to their myelination. This is also said to be the case as regards premature birth.

It is natural that, under such conditions, the pyramidal tracts should atrophy or degenerate (Fig. 312), or should simply remain undeveloped. Those of the other side may become hypertrophied in compensation—according to Dejerine (*R. n.*, 1902), Marie-Guillain (*R. n.*, 1903), d'Abundo, Oeconomakis (*l.c.*), Catola (*cf.* also Fig. 312). The secondary degeneration caused by

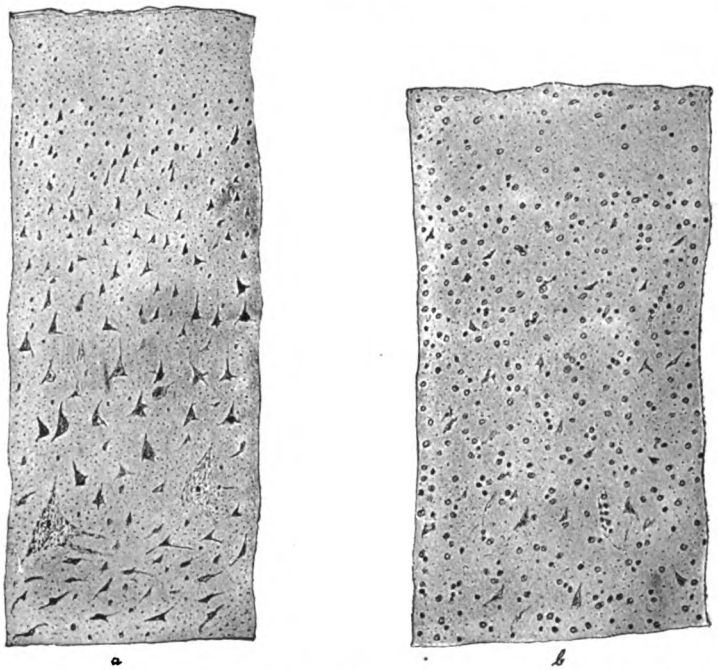

FIG. 315.—*a* and *b*. Ganglion cells of the motor cerebral region: *a*, in the normal brain; *b*, in microgyria. (Oppenheim, Nissl's stain.)

lesions of the cortex may also appear at other places, *e.g.* in the fillet, in the various tracts of the cerebral peduncles, etc. Extensive atrophy of one cerebral hemisphere is often combined with atrophy of the opposite half of the cerebellum, as has been established by previous investigations—specially those of Mott and Tredgold (*Br.*, 1900), and also those of Marinesco, who ascribes atrophy of the cerebellum to that of the basal ganglia.

Symptomatology.—The initial stages can, of course, be studied only in those cases in which the disease commences after birth. In such cases, which may be taken as typical, the usual characteristics are as follows: the child, hitherto healthy, in its first year or from one to three years old, becomes ill with *fever*, *vomiting*, *stupor*, *delirium*, and *convulsions*, which are general or from the first unilateral. In association with these, or after repeated attacks of convulsions, paralysis occurs in the form of *hemiplegia*.

The initial stage lasts from one to several days, but it may extend

to weeks, and in rare cases it is altogether absent. Paralysis then occurs suddenly (as in apoplexy), or in combination with general or unilateral convulsions. The beginning of the paralysis is seldom separated from the stage of convulsions by any considerable interval of time.

It may, however, be actually present at birth, and may be observed either immediately or after some weeks or months—becoming more and more evident as voluntary movement develops on the normal side.

In typical cases, after the lapse of some weeks or months, a progressive improvement sets in. A certain amount of mobility reappears, so that the individuals are able to learn to walk, and to use their arms for the coarser movements. Subsequently, however, very characteristic deformities occur which are due to *muscular contraction* and *spasmodic muscular action*. These must be considered more particularly.

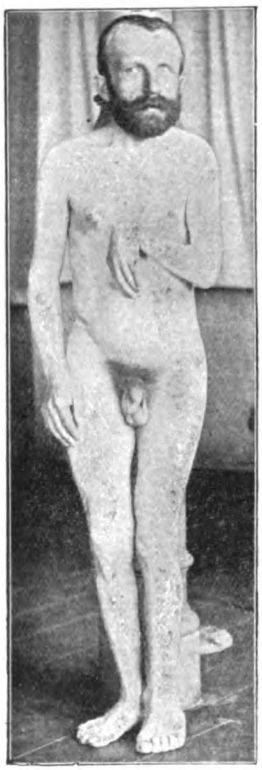

FIG. 316.—Case of infantile spastic hemiplegia of the left side. (Oppenheim.)

If we first of all consider its distribution, we find that the paralysis, like the hemiplegia of adults, affects the arm, leg, facial nerve, and hypoglossus of one side. The tongue is least often involved. The muscles of the face are also only slightly affected as a rule. In repose there may be no asymmetry, but it becomes evident on smiling or weeping. Signs of motor irritation in the face are usually more clearly marked than the paralysis. The arm is as a rule more completely paralysed than the leg.

As the mobility returns, or even earlier, a new derangement becomes visible, viz., muscular rigidity or *contracture*. This may be persistent, *i.e.* it is always present to the same extent and keeps the limb fixed in certain positions: the upper arm is drawn into the trunk, the elbow is flexed, the wrist markedly flexed (Fig. 316) or over-extended, the fingers flexed at all the joints and clenched into the hand, or extended at the interphalangeal joints and over-extended to the point of sublaxation. The leg is usually slightly flexed at the knee-joint, and the foot is in the "pes equinus" position. There may, however, be merely a *spastic innervation of the muscles* in voluntary movement, of such a kind that every impulse to movement leads to tonic spasm, *i.e.* to spasmodic movements instead of single ones. As a general rule a certain muscular tension is constantly present. This increases with any attempt at voluntary movement and constitutes a serious impediment to it. In fact there are certain forms of cerebral infantile paralysis in which stiffness is the only symptom, and paralysis can hardly be said to be present.

Bechterew (*N. C.*, 1900) has given the name hemitonia to this well-known condition. Babinski (*R. n.*, 1904) describes a case in which he considers the unilateral spasms to be the primary condition and the hemiplegia or hemiparesis to be secondary to them.

The exaggeration of the deep reflexes corresponds to the increase in the muscular tonus: an increase in the knee-jerk can always be demonstrated, while ankle clonus is less constant. The Babinski sign is frequently found, and sometimes, though not always, the Oppenheim sign. The degree of tension is by no means the same in all the paralysed muscles: it may be more noticeable in the leg than in the arm; it may be very marked in the muscles of the shoulders, whilst the hand and fingers can be freely moved to and fro—sometimes even beyond ordinary limits, so that the fingers may be considerably over-extended at the metacarpo-phalangeal and inter-phalangeal joints (Fig. 317). Details regarding the position of the hand and fingers are given by W. König.

A further development, which is more general in infantile hemiplegia and occurs to a much greater degree in children than in adults, is that of *associated movements*, which are usually present and often well marked. They are rarely so pronounced as in the cases described by Westphal, in which the associated movements corresponded roughly in extent to those of the extremity which was voluntarily moved. I have found them most frequently in the extensor hallucis longus, which contracted with every step —sometimes with every movement of the arm.

FIG. 317.—Hyper-extension of the thumb from athetosis in infantile spastic hemiplegia. (Oppenheim.)

Even more characteristic of spastic infantile paralysis than the above-mentioned signs are those of motor irritation which have been already described under the terms *athetosis* or hemiathetosis and *hemichorea* (*cf.* p. 690). The latter signs are present in most cases—in some they are only suggested, but in others they constitute the main symptoms. Athetosis is more frequent than chorea, but the points of difference are not very serious. In one case chorea was present in the upper extremity and athetosis in the lower. It may exist from birth in cases of congenital paralysis, or develop as primary athetosis in extra-uterine life, without being preceded by paralysis. As a rule it is found either earlier or later, combined with hemiplegia in the stage when voluntary movement is again partly re-established. It may be continuously present—although in varying degrees of intensity—or may accompany only voluntary movements, or be brought on specially by mental excitement. I have once seen in a case of this kind that every stimulation was followed by a tonic extension in the arm and leg of the paralysed side, and by spasmodic movements of the eyeballs and head, contraction of the muscles of the face and the tongue. The movements of the hand and fingers are specially involved in athetosis: the patient can only open his closed hand with diffiulty, or shut his open hand very slowly, etc.

A variety of tremor allied to intention tremors occurs in very rare cases. Ataxia is described by König and by Bouchaud (*R. n.*, 1904). Apraxia was observed in one case by Cassirer and Oppenheim.

There are cases in which there is complete recovery from the paralysis and slight athetosis—*i.e.* a slight inclination to associated movements is all that remains from the earlier disease.

Headache and sensitiveness on percussion of the scalp at a given spot were present in some of my cases.

Sensation is for the most part preserved; hypæsthesia has been observed only in exceptional cases (Oulmont, Gaudard, Raymond, König). I have noted it in a recent case only four weeks after the onset of the paralysis, and once in an adult (who had suffered from cerebral infantile paralysis), in combination with hemiathetosis. Here the functions of the special senses were also affected on the same side, so that a combination with hysteria could not be entirely excluded, and indeed vaso-motor disturbances were also present. In several cases I was able to show that although the sensibility of the skin was unaffected, Wernicke's so-called astereognosis (*cf.* p. 704) or tactile paralysis was present. As, however, the hemiplegia was congenital, or at least dated from earliest childhood, one had to bear in mind the fact that the patient had never acquired any tactile memory pictures in relation to the paralysed hand. This view has been accepted by Dejerine, Claparède, and others. It did not apply, however, to two of our cases, in which the previously paralysed hand was used for all purposes. Ossipow [1] has noted in a few of our patients disturbance of the stereognostic power of perception. A complaint is sometimes made of great pain in the affected side of the body.

Hemianopsia was observed in only a few rare cases (Freud, Oppenheim).

Not infrequently the hemiplegia (especially when on the right side) is associated with aphasia, which usually sooner or later entirely disappears. As a rule it is a motor aphasia. If the child had not reached the stage of being able to speak, the development of his power of speech would be delayed by the occurrence of hemiplegia. Dysarthria and other disorders of speech also occur.

An infrequent complication which should be mentioned is a paralytic affection of the muscles of the eye (Menz, Luce, and others). Loss of the pupil-reflexes (König, Oppenheim, Ossipow) and nystagmus seldom occur. Atrophy of the optic nerve may also appear (König, Collier: established anatomically by Bourneville and Schwarz; see supplement to this chapter).

The paralysed limbs all show a similar arrest of development; and the muscles have *decreased in size* (without any qualitative change in their electrical excitability).

Kellner (and Oppenheim) have been able by means of radiography to establish the existence of changes in the bones of the paralysed limbs. W. König (*Z. f. N.*, xix.) discusses the relation of these hypoplexias to the cerebral process. Slight hemiatrophy of the facial muscles has occasionally been observed. In a case in which hemianæsthesia was also present, there was ulceration of the leg, which I diagnosed as neurotrophic. At the same time, in view of the extreme rarity of this occurrence, we must bear in mind the possibility of a combination of cerebral and spinal disease (gliosis, etc.).

Muscular hypertrophy is sometimes associated with athetosis. Changes in the bones of the skull, depression, formation of fissures (porocrania), and decrease in circumference are inconstant symptoms (Peterson and Fischer, Breschet, Meschede, Bergmann, and others).

See on this subject also Bourneville ("Compt. rend. des enf. idiots," etc., Bicêtre, 1903), Boncour (*Arch. de Neurol.*, 1904).

In the case of a girl suffering from right spastic infantile hemiplegia I observed a defect in

[1] *M. f. P.*, viii.

the left frontal bone, over which the cephalic bruit was clearly audible; in the same case I found telangiectasis of the nose. Kalischer (*N. C.*, 1899) has made preparations from a case in which a child suffering from spastic infantile paralysis had telangiectasis of the scalp and an analogous new-formation on the surface of the brain, particularly in the membranes over the motor zone. Similar observations have been made by Lannois-Bernoud (*Nouv. Icon.*, 1898), Strominger (*Spitalul*, 1901), Cassirer (*N. C.*, 1902).

Atrophy of the testicle, abnormal smallness or even hypertrophy of the mamma (Oppenheim, Lannois), a fixed backward position of the small and ring fingers (Féré), malformations of the eyes (microphthalmia, buphthalmus), and other arrests of development on the paralysed side are occasionally found. Wachsmuth (*A. f. P.*, Bd. xxxviii.) also describes interesting malformations.

In several cases of this kind I confirmed the complete absence of the sexual functions.

The most important of the other symptoms of cerebral infantile paralysis are *imbecility* and *epilepsy*. Epilepsy is not always present but it is so in a large percentage, probably in half or even two-thirds of the cases. It may develop simultaneously with the first convulsions, or after an interval of months, years, or even decades. Usually a period of from one to two years elapses between the onset of the hemiplegia and that of epilepsy. In its character the epilepsy rarely corresponds in all particulars with true epilepsy—as has been specially pointed out by Wuillamier and Wachsmuth. The fits are usually confined to the paralysed side, or at least they commence and are more strongly marked in it. They correspond closely to the picture of *cortical* epilepsy. If they are confined to one side, consciousness may be retained. The initial cry, the biting of the tongue, and the loss of control of the sphincters are usually absent. Altogether the attack is not as a rule so completely developed as in true epilepsy. None the less almost every variation which has been observed in attacks of true epilepsy occurs here also. The attacks, however, do not often reach such an intensity, and mental disturbances are less prominent (W. König).

The individual attack either begins spontaneously or is brought on by mental excitement.

Thus I had under my care a girl suffering from cerebral infantile paralysis, in whom an attack could be brought on at will by fright—*e.g.* by a chair being allowed to fall to the ground behind her back. According to the definite statement of her father, an attack on the point of development could sometimes be prevented by a counter-shock.

These convulsions may, owing to their frequency and intensity, be very distressing. The status epilepticus also occurs. There are cases of cerebral infantile paralysis in which the symptoms of paralysis entirely disappear, leaving only the epilepsy, which had developed either simultaneously or later, to indicate, by the appearance of twitching in the side formerly paralysed, or by a temporary paralysis succeeding an attack, etc., the previous existence of the disease.

In a large proportion of cases the mental development of the child suffers, and every transition occurs between slight peculiarities of character and weakness of intellect to complete *idiocy*. Intense irritability and maniacal conditions are not uncommon. Mental weakness is most fully developed in cases combined with epilepsy. The manifold relations between idiocy and cerebral infantile paralysis have been described by Bourneville and König and by Wachsmuth.[1]

[1] *A. f. P.*, xxxiv.

The intelligence may, however, remain unimpaired: indeed, I know a lady suffering from this disease, who has passed most brilliantly her final school and teachers' examination.

An attempt has been made, especially by Freud (as also by Osler, Rosenthal, Oddo, Collier, Hascovec, and others), to classify the different cases of cerebral infantile paralysis and to give greater precision to the characteristic features of some of them. It is certainly quite natural to distinguish congenital forms from those appearing in extra-uterine life—but apart from the fact that there are no striking clinical characteristics it is quite impossible to prove whether the disease should be regarded as congenital or acquired. Up to a certain point, however, the classification may be carried out. In cases of congenital origin the symptoms appear soon after birth or within a few months. They have at first (during the first few years) a progressive character. At one time the paralysis is most conspicuous, at another the signs of motor irritation. The child learns to walk late and is slow in learning to speak. Very frequently all four limbs are affected (see below).

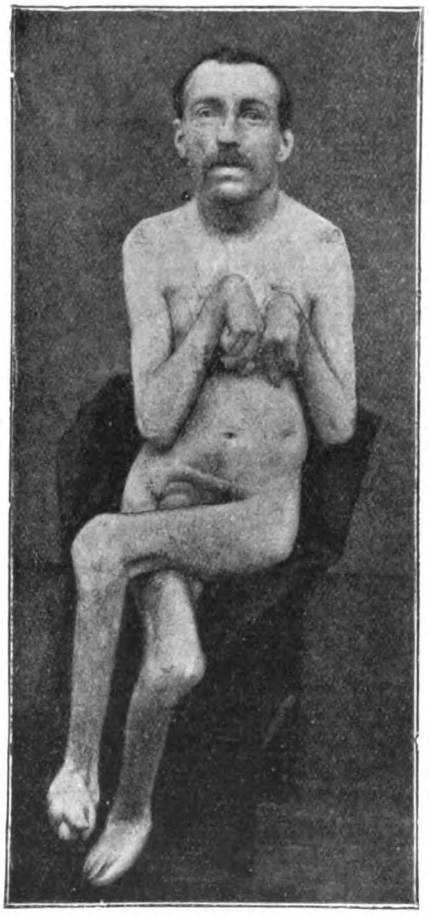

FIG. 318.—Spastic diplegia with contracture of all four extremities. (After Dejerine.)

Strümpell has sought to differentiate one form, which he regards as analogous to spinal paralysis (see above). In this form the disease is always *acquired* in earliest childhood. There is a definite initial stage resembling an acute infection, followed by convulsions and hemiplegia, and more or less degree of recovery with improvement in the power of movement, within a few months: later symptoms of motor irritation, very frequently of epilepsy appear.

Freud has pointed out that there are cases in which the disease develops gradually and does not begin with paralysis but with chorea or athetosis. This form, named by him choreic paresis, is further distinguished by commencing, not in earliest childhood but from the age of three to six years, and by the fact that aphasia, epilepsy, and dementia are usually absent, and that contracture is less marked, whilst chorea or athetosis form the prominent symptom.

Further divisions may be made according to the *extent* of the paralysis and the contracture. There are cases in which the rigidity and weakness extend to the leg on the healthy side. Athetosis often appears—though in a lesser degree—in the limbs of that side. These cases form the transi-

tion to an important group (specially studied by Freud) in which the hemiplegia is really bilateral and all four extremities are affected by paralysis and spasms (*diplegia spastica infantilis*).

Freud groups cases of this kind under four headings: 1, general cerebral rigidity (Little's disease; but he restricts this conception too much); 2, paraplegic rigidity; 3, bilateral hemiplegia; 4, general chorea and bilateral athetosis. This division, however, as he himself allows, cannot be rigidly adhered to: on the contrary all kinds of transition and mixed forms occur. The type mentioned under 1, and especially that under 2, have already, on account of their spinal character, been considered in another place (p. 181).

As a rule rigidity and weakness are most prominent in the legs, whilst choreic-athetotic movements predominate in the upper extremities. Sometimes, however, general athetosis or chorea is present. Bilateral

FIG. 319.—Facial expression and attitude in athetotic-spastic diplegia. (Oppenheim.)

athetosis may prepare the way for a progressive disease which appears in later life (Albutt, Oulmont, Andry, Lewandowsky, *cf.* p. 694). The intelligence may, as already mentioned, remain quite unimpaired: in this form I have proved this in several instances. Marie, however, certainly goes too far when he declares it to be the rule that the mind remains intact in bilateral athetosis. Epilepsy is less frequently present in these forms of diplegia. In many cases of spastic infantile diplegia the power of articulation is also lost: thus I have repeatedly found distinct signs of glosso-pharyngo-labial paralysis (see corresponding chapter) with dysarthria and even with dumbness in these complicated cases of bilateral infantile paralysis. Aphonia or dysphonia, caused by spasm of the adductors, may also be a symptom of this disease. Habitual subluxation of the lower jaw also occurs.[1] Since I have written on the subject

[1] These phenomena, however, are frequently found in healthy persons: they are possibly to be looked upon as stigmata of degeneration.

Bouchaud,[1] König, Collier, Zahn,[2] Variot-Ray, and Peritz [3] have all described cases of this bulbar or pseudo-bulbar variety. With regard to a few reflex movements which occur in this disease (reflex feeding movement or movement of the gums and lips, etc.), compare the chapter on infantile pseudo-bulbar paralysis, p. 673. The expression of the face and the whole aspect in cases of this kind are very characteristic (Fig. 319 and Fig. 320). The difficulty in speech may also be caused by the fact that the muscles pertaining to articulation and respiration are involved in the spasms or in the choreic twitchings. It is also of interest to note that in this form the muscles of the trunk, and especially those of the neck,

FIG. 320.—Daughter of patient shown in Fig. 319, suffering from the same disease. (Oppenheim.)

are often affected so that children cannot hold up their heads. (This feature is absent in the cases illustrated by Figs. 319 and 320. It may readjust itself in later life.) All these points combine to produce a picture so typical that the affection may be recognised at the first glance, and I have often been able to diagnose the case as the child was being carried in.

A remarkable symptom in these cases, and one which occurs in most of them in my experience,[4] is *abnormal timidity*, *i.e.* violent starting at the slightest sound. In one of my patients this was so marked that the child, on hearing the noise of boiling water in the kitchen, not only quivered through its whole body, but became asphyxiated and faint.

[1] *Rev. de Méd.*, 1895.
[2] *M. m. W.*, 1901.
[3] "Pseudobulbär- und Bulbärparalysen des Kindesalters," Berlin, 1902.
[4] *M. f. P.*, xiv.

The phenomenon is, in the first place, to be ascribed to the fact that with the loss of the motor zone the inhibitory centres are also thrown out of gear, but more particularly to the circumstance that now that a large part of the cortex is no longer capable of function, stimuli reaching the brain through the sensory nerves strike the sub-cortical and bulbar centres, etc., with proportionately greater force. It is due, therefore, less to an increased mental excitability than to an *exaggerated acoustico-motor* reaction.

The majority of cases of diplegia are congenital or arise during labour, and general rigidity (in Freud's sense) is for the most part a paralysis due to labour (abnormal position, contracted pelvis, asphyxia neonatorum). The paraplegic rigidity is very often the result of premature birth. It is obvious that these phenomena correspond to a bilateral disease of the hemispheres. With the exception of foci of inflammation and of softening, porencephalies, bilateral meningo-encephalitis with its consequences (Massalongo), it would seem that the cause of this particular form is comparatively seldom a grave disease of the centres, but rather a primary atrophy or an arrest in the development of the cortical nerve elements, viz. the ganglion cells and nerve fibres—as Collier has found from his investigations. When the patient who is shown in Fig. 319 died this year, no essential change in the brain could be detected by the naked eye, and no microscopical investigation has as yet been undertaken. One would not expect that the changes in the *cord* found in individual cases of this kind (Eisenlohr, Dejerine) are at all common.

Diagnosis.—The differential diagnosis does not present any great difficulty. It is hardly possible to mistake it for *spinal infantile paralysis* : the spastic and non-degenerative character of the paralysis, its combination with athetosis, chorea, etc., and involvement of the facial group of muscles form striking marks of distinction. A few observations, however (such as those of Lamy, Beyer, Oppenheim, Marie, Rossi [1]) point to the possibility of the two affections being combined in one individual.

The diplegic form is also readily distingushed by one who has seen several cases of this kind. I have indeed seen some cases which on superficial examination suggested simple chorea, but its early commencement (soon after birth), its persistency, and the spastic weakness of the legs, which usually becomes very apparent as the disease progresses, soon pointed to the correct diagnosis.

In the very rare cases of monoplegia of the arm—whether from the onset confined to that limb, or remaining from a hemiplegia—there is certainly a resemblance to the paralysis incident on delivery, but the latter, if it persists at all, is merely flaccid paralysis.

It has been definitely proved that this disease may be marked by the symptoms of epilepsy, the paralysis and other symptoms remaining entirely in the background, whilst the convulsions persist. But the character of the epilepsy, the history of the case, the signs of slight athetosis or of marked associated sympathetic movements often make it possible to recognise the disease.

The actual pathological process cannot always, in individual cases, be diagnosed. Even porencephaly, as Felsch (*A. f. P.*, xxxvi.) rightly states, cannot be recognised with certainty, in spite of the attempts made by Brissaud and Sommer. There is the same difficulty with lobar sclerosis, which Bischoff has tried to define more precisely. More information on this point will be found in Sommer (*M. f. P.*, xv.) and Dannenberger (*l.c.*).

[1] *Nouv. Icon.*, xx.

Prognosis.—Cerebral infantile paralysis is an affection with very slight tendency to recovery. There are certainly some cases which recover to such an extent that they may be regarded as almost completely cured: a certain awkwardness in one hand, a slight tendency to athetoid and associated movements, or possibly some condition resembling epilepsy, may be the only persistent symptoms of cerebral disease. In the great majority of cases, however, the disease is incurable, and the improvement only reaches a point where the paralysed limbs attain once more to a certain measure of mobility, which remains incomplete owing to the contracture and athetosis. The latter may render the extremities entirely useless, the finer movements of the hand and fingers being quite impossible. It is hardly conceivable that contracture, athetosis, and chorea, when once developed, can ever entirely disappear. Improvement in the paralytic symptoms is almost confined to the first year, but may continue to progress for years.

This description of the prognosis refers to completely developed cerebral paralysis, not to the acute disease which is its cause, and we must not forget that some of the processes mentioned (the hæmorrhage and the acute encephalitis) may completely disappear.

There is always some fear that epilepsy may develop; if two to three years have elapsed without convulsions having appeared, they are less likely to do so, but they have been known to develop after ten years or longer. With advancing years the fits occur more rarely and are said to disappear completely in the fifth decade (Bourneville, Wachsmuth). Yet death during the "status epilepticus" may occur at any time. It becomes evident during the very first years whether the mind is to be seriously affected; but, without further signs, it is not necessary to conclude from a mere delay in the development of speech and intellectual power that there is permanent defect. Aphasia is a symptom of good omen—it almost always disappears.

The patients may live to a very great age and are sometimes, up to a certain point, capable of work. Thus one of my patients is a carrier: he uses the affected left hand only for rough work, whilst he does his real work with his right. Another patient acts as a newspaper-deliverer, etc. The lady whom I have already mentioned is a successful student of natural science. Collier, however, asserts that spastic athetotic diplegia is usually progressive, and that those suffering from it die young, seldom living beyond the age of puberty. That is not my experience, however, and Londe (*R. n.*, 1901), for example, instances a woman fifty years of age, who had suffered from this disease from the first year of her life.

A recurrence of the primary disease has rarely been observed.

The prognosis of Little's disease is comparatively favourable. In it the functional disturbance may gradually undergo a distinct improvement, which, however, never amounts to complete cure.

Treatment.—We seldom have an opportunity of treating this complaint at its onset. The early symptoms, as a rule, require antiphlogistic treatment, *e.g.* the use of ice-bags, local blood-letting, etc. Compare, however, the previous chapter. Surgical treatment is recommended by Cushing,[1] especially for birth-paralyses. He has succeeded several times in obtaining a satisfactory result by means of trephining and the removal of the accumulation of blood in the meninges.

[1] *Amer. Journ. of Med. Sc.*, 1905.

Our therapeutic efforts should for the most part be directed to the paralysis, contracture, athetosis, and epilepsy. The effects of treatment are certainly not striking: yet, in certain cases, one may expect some improvement from the use of the *faradic* or *constant current*, massage, gymnastics, and *passive exercises*. I have seen one case in which very energetic parents, under the mistaken impression that lack of will-power was the cause of the paralysis, succeeded in obtaining considerable improvement by constantly insisting upon the use of the disabled hand. Hoffa holds that he attained good results in this condition by the use of splints and a pelvic girdle to correct the rotation and the pointing of the toes. I have personally found little benefit from such apparatus in spastic hemiplegia and diplegia. The epilepsy requires systematic administration of *bromide preparations*, but the effect cannot be relied upon. To lessen the chorea and athetosis Sachs recommends that an iron plate should be carried in the restless hand.

In many cases (Eulenberg,[1] Hoffa,[2] Rochet, Vulpius, Codivilla,[3] and others) plastic operations on the tendons are said to have greatly lessened the disturbance of function caused by the contracture and paralysis.

Hoffa, for example, to neutralise the flexed and pronated condition of the contractured upper extremity, cut the biceps tendon and the bicipital fascia, then detached the pronator teres from its origin at the internal condyle, pushed it to the other side between the supinator brevis and the flexor muscles, and stitched it to the external condyle above the supinator brevis.

Wittek's[4] statement that transplantation has a favourable effect on the choreic-athetotic movements is of great interest. I have seen a very good result in one case. This question is treated in more detail by J. Fränkel. Spiller-Frazier[5] have lately strongly recommended this course of action, specially the use of nerve-anastomosis (greffe nerveuse, see p. 415), in these conditions—at least in cases where athetosis is limited to particular areas of nerves and muscles.

In view of the hopelessness of symptomatic treatment any attempt to influence the disease directly should be warmly welcomed. This applies to the efforts of *surgical* treatment, which is now directed to this class of cerebral diseases. As meningeal thickenings, or meningeal and intra-cerebral cysts are found in some cases, it is conceivable that excision of the thickened portion of the meninges and emptying of the cyst may remove some noxa which has been a permanent source of irritation. It was hoped that in this way it might be possible to improve epilepsy. Certainly up to the present time the results obtained by surgical interference in cerebral infantile paralysis are not of a character to encourage fresh efforts, but certain writers (Starr, Sachs, Gerster, Bullard, Ballance, Chipault, and F. Krause[6]) have quite recently advised this form of treatment and have laid down definite indications for it. The views of Rahm[7] and Amberger[8] on this point are also worthy of attention. In the case described by the latter Röntgen rays were of the greatest service.

In one particularly severe case of this kind in which the violence

[1] *D. m. W.*, 1892.
[2] *Z. f. physik. Therap.*, vi.; *D. m. W.*, 1906; further, J. Fraenkel, *Z. f. orthop. Chir.*, xv.
[3] *Revue d'Orthopéd.*, 1900, and *Arch. di Ortoped.*, 1902.
[4] *Mitt. aus d. Grenzgeb.*, xii.
[5] *Journ. of Nerv.*, 1905, and *Contributions, Univ. of Penn.*, ii., 1906.
B. k. W., 1903 and 1905. [7] *Inaug. Diss.*, Zürich, 1896. [8] *D. m. W.*, 1906.

and frequency of the epileptic attacks made existence almost insupportable, I was, several years ago, induced to resort to operative treatment.

A girl, twelve years of age, had suffered since her fourth year from hemiplegia and hemiathetosis on the right side. These were accompanied by convulsions which affected the right side of the body. The skull was sensitive to percussion over the left parieto-temporal region. Sonnenburg trephined at this part and found a cyst, the size of a plum, in the meninges. This was evacuated without any injury to the brain. The wound healed well, and at first the fits were less frequent and violent. The athetosis also seemed somewhat lessened. But the improvement did not last long and, after the lapse of a year, the patient died in status epilepticus. The post-mortem examination revealed the existence of an extensive area of softening—involving the central convolutions and the lower part of the parietal lobe (probably of meningo-encephalitic origin)—and also atrophy of the entire left hemisphere. It is natural that, in the presence of such modifications, the emptying of the cyst could have little effect.

On the strength of this experience and on mature deliberation, I have since then subsequently opposed surgical interference, and I have in consequence found myself several times in conflict with my colleague Krause. On the other hand a short time ago I felt it to be my duty once again to recommend an operation in a case in which the monoplegic character of the paralysis and the signs of irritation pointed to the process being superficial and of slight extent. As I had expected, two cysts of the size of a nut were discovered in the sub-cortical white matter of the leg centre. The operation gave promise of a satisfactory result, but the girl came from Russia and it was therefore impossible to keep her under observation. Chipault (*R. n.*, 1898) claims to have influenced epilepsy favourably, in a few cases at least, by the removal of cicatrices and by the excision of thickened meninges.

We have as yet insufficient proof as to the effect of thiosinamin or fibrolysin on the scars which result from these processes; an observation of my own—in which it was at first successful—is still incomplete.

This is not a fitting opportunity to discuss the cranial operation recommended by Lannelongue. At the same time we may point out that the results, according to the experiences of Keen and Spiller and the digest of Pilcz, appear to have been distinctly unfavourable, so that surgical interference can no longer be regarded as more than barely justified.

Appendix

There are other hereditary affections of the nervous system which appear to stand in close relationship to the diplegia just described, although in many respects they correspond to a certain type of spinal disease.

The majority of these observations refer to the occurrence of spastic rigidity of the limbs, either congenital or appearing in early childhood in the case of several children of one family (Schultze, Bernhardt,[1] Kojewnikoff, Newmark,[2] Erb, Krafft-Ebing, Jendrássik, and others). Thus the upper extremities may be attacked and cerebral symptoms—particularly strabismus, disturbance of speech, and nystagmus—may appear as signs of the disease (as observed by Pelizaeus [3] and others).

Sachs [4] has described another type—which should perhaps be included here—observed almost exclusively amongst Jews, of which the chief signs are: idiocy, spastic (or possibly only atonic) paralysis of all four extremities, blindness from atrophy of the optic nerve with a characteristic change in the macula lutea, which forms a red spot surrounded by a white ring. This type had been previously observed by Tay and others. The children at first develop normally and then—when a few months to a year old—are attacked by the disease. Characteristics other than the

[1] *V. A.*, Bd. cxxvi.
[2] *Amer. Journ. of the Med. Sc.*, 1893, and *Z. f. N.*, xxvii.
[3] *A. f. P.*, xvi.
[4] *Journ. of Nerv.*, 1903; *D. m. W.*, 1903.

sight affection are the rapid progress of imbecility and spastic paralysis, and the marasmus which develops and soon causes death. Nystagmus and salivation are sometimes additional symptoms. Degeneration of the cortical cells, especially the pyramidal cells, has been found, but Sachs is convinced that the term "agenesis corticalis" does not completely cover the pathological process, as a degeneration of nerve cells in the sense of Gowers' abiotrophy due to an arrest of development is also present. He therefore suggests the name "amaurotic family idiocy" to correspond with the clinical appearances. Kingdon, Russell, and in particular Frey have undertaken further investigations with regard to the pathological process. This author noted very extensive changes in the brain and spinal cord, not only in the cells of the latter, but particularly atrophy of the fibres, *e.g.* in the pyramidal tracts. In opposition to Sachs he maintains that the disease begins only after birth. Schaffer [1] shares this view. He, however, assumes that there is also an inherited weakness of the central nervous system, especially of the ganglion cells, and a premature degeneration, due to the exhaustion caused by this. He has found atrophy of the cells not only in the brain, but in the sub-cortical ganglia, the medulla oblongata, etc.

The views advanced by Alzheimer (*N. C.*, 1906) and by Poynton-Parsons and Holmes (*Br.*, 1906) on this point are worthy of consideration. Further observations of the same kind have been made by Peterson, Hirsch, Holdau, Falkenheim, S. Kuh, Higier,[2] Mühlberger [3] (whose investigation, however, carries no weight on account of the absence of the macular lesions), Heveroch (*D. m. W.*, 1904), Gross (*N. C.*, 1905) Spielmeyer (*N. C.*, 1905), Sterling (*N. C.*, 1906), Huismans (*D. m. W.*, 1906). The hyperacusis mentioned by Higier would probably be covered by the accentuated acoustico-motor reaction, which exists, as I have shown, in certain forms of diplegia (*q. v.*). Russell describes the affection as "infantile cerebral degeneration." Amongst recent contributions to this question those of H. Vogt (*M. f. P.*, xviii., with biblio.) are of special importance; they refer also to a juvenile type of the disease. I should mention also Mott's pathological studies (*Arch. of Neurol.*, iii.).

I have observed a combination of spastic-ataxic paralysis with talipes cavus in several members of the same family, and a short time ago in an individual whose parents were related to one another, and who was himself a twin. I observed a combination of congenital spastic paralysis with talipes cavus—a consequence of atrophy of the peronei—and prognathism and nystagmus. Atrophy of the optic nerve also developed but only in later life. The various possibilities are by no means exhausted with these mentioned. Trénel describes a family disease, of which the chief symptoms are epilepsy, dementia, and spastic paresis. A similar type was described by Pesker and by Bourneville-Crouzon, while a family affection described by Clarke is again quite distinct from this type.

We have as yet no very reliable information as to the pathological process which underlies all these conditions, but, by their symptoms, they are evidently so closely related to diplegia that it is impossible to draw a sharp line of distinction between them. On the other hand they are nearly allied to the so-called hereditary form of spastic spinal paralysis (see p. 179).

The neurone theory is a sufficient indication that too much importance should not be attached to the distinction between these cerebral and spinal affections of the motor system.

[1] *N. C.*, 1904, 1905; *Journ. f. P.*, vi.; *A. f. P.*, Bd. xlii.; *Journ. f. P.*, x.
[2] *Gazet. lek.*, 1901.
[3] *M. m. W.*, 1903.

Brain Abscess

For literature see: Macewen, "Pyogenic Infective Diseases of the Brain and Spinal Cord," etc., Glasgow, 1893; O. Koerner, "Die otitischen Erkrankungen der Hirns," etc., 3rd edition, Wiesbaden, 1902, and "Nachträge zur dritten Auflage," Wiesbaden, 1908; H. Oppenheim, "Der Hirnabszess"; Nothnagel's "Spez. Path. und Ther.," Bd. ix., Teil I., 3. Abt., in two editions.

See also the surgical works of Bergmann, "Die chirurg. Behandl. d. Hirnkrankheiten," Berlin, 1899; Chipault, "Chirurgie opérat. du syst. nerveux," 1894; Broca-Maubrac, "Traité de Chirurgie cérébrale," Paris, 1896; Ballance, "Some Points in the Surgery of the Brain," London, 1907; Barnhill and others, with Discussion, *Journ. Amer. Med. Assoc.*, 1905.

Accumulation of pus in the brain substance is the result of a *purulent encephalitis* produced by *micro-organisms*. It may be of *traumatic* origin, or the infective matter may come from *suppurative foci* in the immediate *neighbourhood* of the brain or at more *distant* sites. It is only in rare cases that the septic material circulating in the blood passes directly to the brain and gives rise to a primary purulent encephalitis.

In a great percentage of cases the abscess is due to *trauma*. There are always open wounds on the skull, and even an injury of the soft parts may form the access through which the inflammatory irritant finds its way into the brain substance. In most cases, however, there are complicated fractures or cut-wounds of the brain, followed by protracted suppuration, and in many foreign bodies have entered the brain. *Simple contusion of the skull cannot produce brain abscess* (Bergmann).

We must of course remember that heavy pressure upon the skull may cause a fracture at its base which communicates with the nasal cavity or with its accessory cavities, and that the micro-organisms may penetrate into the brain from this point. This is on the whole a very rare occurrence. The fact, demonstrated by experiment, that micro-organisms in the blood are apt to choose as the site of their settlement a point at which there has been some traumatic lesion of the brain (Ehrenrooth), is not of great practical importance.

The formation of the abscess takes place immediately after the injury, or a few days later; in such cases there are usually superficial cortical abscesses, which, on account of their frequent association with meningitis, are not of special clinical interest. Or there may be an interval of comparative or complete health; between the injury and the suppuration in the brain the abscess in this case usually develops within the brain, in the medullary substance, and usually in the segments beneath the parietal or frontal bone, as this part of the skull is most frequently injured. It should always be remembered that the *trauma may be slight and forgotten* long before the symptoms of brain abscess appear. Cases have been observed in which there was an interval of 10 to 20 and even of 30 years between the injury and the onset of the cerebral symptoms.

Suppuration on the skull very often provides the septic matter which passes into the brain. *Chronic purulent otitis media* is the most important cause of brain abscess, as it gives rise to a half, or at least a third of all the cases. This otitis is usually acquired in childhood—especially after acute infective diseases. It may exist for many years before the brain becomes involved. This danger arises from suppuration of the tympanic cavity and its accessory cavities—especially the cells of the mastoid process—from caries of the petrous bone, and especially from *cholesteatoma*. The tympanic membrane is almost always perforated, and a discharge of pus from the ear is, or has been present some months or years previously.

Exceptions to this are very rare, but Kölpin, for instance, has lately described a case of brain abscess without perforation of the tympanic membrane. Danger arises specially from acute returns of chronic suppuration and blockage of the discharge by granulations. It is only in a small number of cases—although according to recent experience these are increasing—that the inflammation of the brain follows an *acute otitis media* (with or without perforation of the tympanic membrane), but the suppuration of the ear may have disappeared before the brain symptoms become evident.

Heimann collected from the literature 457 cases of abscess due to *chronic*, and 113 due to *acute* suppuration of the ear (*A. f. Ohr.*, Bd. lxvi.).

Brain abscess may also be caused by caries of the mastoid process in diabetes (Kuhnt, Körner).

The parts of the brain adjacent to the bony walls of the organs of hearing become infected. The temporal lobes are infected from the superior part of the tympanic cavity, from which it is only separated by a thin lamina of bone, or from the roof of the mastoid antrum. The cerebellum is infected either from the mastoid process, the cells of which are usually involved if there is suppuration in the cerebellum, or from the labyrinth. The bone lesion usually extends as far as the dura (Körner): sometimes a fistula leads from the abscess through the thickened, adherent, or pus-infiltrated meninges to the bone focus, but as a rule it is separated from it by a more or less thick layer of brain tissue. Thus there is not as a rule a continuous propagation of the pus from the bone to the brain substance, but the micro-organisms penetrate through the cortex more or less deeply into the medulla, following the perivascular lymph-spaces, or making their way backwards from the thrombosed veins of the pia into the brain tissue. The pus may also be carried along the *acoustic* and *facial* nerves, and through the vestibular aqueduct (Jansen). In addition to intra-cerebral suppuration, there is often extradural suppuration in the middle or posterior cranial fossa, or at both of these sites, giving rise as a rule to thrombosis of the sinuses. This in itself is a frequent complication of abscess, especially of the cerebellar form.

For details of connections between diseases of the petrous bone and of the brain, see Körner, Jansen, and Barbarin (*Thèse de Paris*, 1902).

Subdural abscess, or localised, encapsulated suppuration of the meninges, which usually also involves the cortex, is on the whole a rare form. It occurs mainly in the middle cranial fossa (Macewen, Jansen, Lucae), but may also occupy the posterior (Heine [1]).

It is a rare occurrence for the brain abscess to arise from carious processes of other bones (ethmoid, sphenoid), from erysipelas of the face or skin of the head, suppuration in the nasal cavity or orbit, infection after the removal of polypi from the nose, etc. Cases of *rhinogenic* brain abscess have lately become much more frequent (Kuhnt, Dreyfuss,[2] Moritz [3]).

As regards a *metastatic* (embolic) origin, it is chiefly purulent diseases of the bronchi and the lung tissue (*bronchiectasis*, *gangrene*, and *abscess of*

[1] *D. m. W.*, 1903.

[2] " Die Krankheiten des Gehirns und seiner Adnexe im Gefolge von Nasenkrankheiten," Jena, 1896.

[3] *Brit. Med. Journ.*, 1905.

the lungs), and empyema, which involve the brain (Virchow, Biermer, Gull, etc.). Here, as in pyæmia, *ulcerative endocarditis*, puerperal infection, etc., we have as a rule to deal with *multiple abscesses*. Nähter[1] found suppurative foci in the brain in eight out of a hundred cases of gangrene of the lungs.

Claytor (*Brit. Med. Journ.*, 1891) collected 58 cases of pulmonary brain abscess; 20 of these were bronchiectasis, 10 empyema, 9 purulent bronchitis, 7 gangrene, 6 tuberculosis, 3 abscess of the lung, and 2 pneumonia. The left side was usually affected. Bouchez treats of brain abscess in pneumonia (*Thèse de Paris*, 1906).

Böttcher has discovered lung pigment in brain abscess. Aphthæ may produce metastatic brain abscess (Zenker, Ribbert), and so may *actino-*

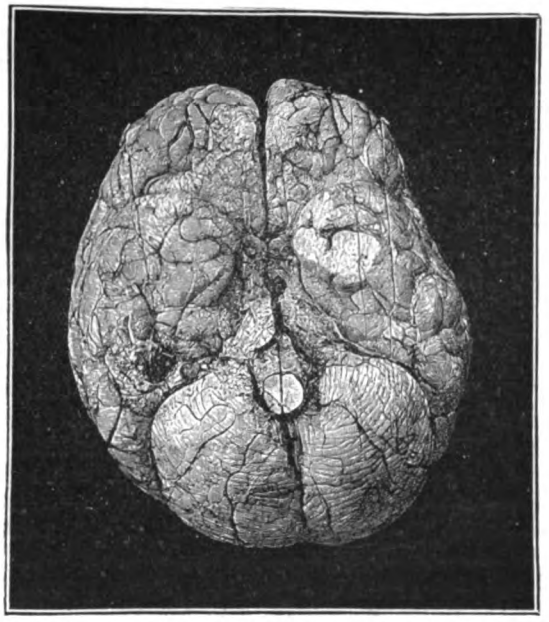

FIG. 321.—(After Macewen.) Localisation of an otitic focus of suppuration in the temporal lobe.

mycosis. In one case of otitis after influenza and pneumonia the pneumococcus was found in the pus of the brain abscess. *Phlegmonosis*, pyæmic and septic processes, may infect the brain from distant parts of the body. The embolism may arise from suppuration of the bones or joints, from a whitlow (Eiselsberg, Dogliotti), a carbuncle (Nielsen), septic endometritis, after a miscarriage (A. Westphal), an abscess of the liver, after dysentery, (Kartulis), suppurating bronchial glands (Ferrari, Schlagenhaufer), perityphlitis (Grawitz), etc. In Hinsdale's case the infection arose from the umbilicus of a new-born child. This mode of origin is, however, exceedingly uncommon.

There are other cases which cannot be attributed to any of these causes. In some of these described as the "idiopathic form of brain abscess," the actual cause, *e.g.* an injury, may have been overlooked. In rare cases

[1] *A. f. kl. M.*, Bd. xxxiv.

the *tubercle bacillus* has been found in the internal wall of the granular tissue lining the abscess, and in the pus itself (A. Fraenkel). I have found this in a case of operation during life. In other cases we must assume that the abscess is related to an infective disease, especially to *epidemic cerebro-spinal meningitis*, the abscess either following or developing at the time of an epidemic (Strümpell). Other infective diseases, such as *erysipelas*, *influenza*, *measles*, *typhoid*, etc., may give rise to purulent encephalitis by means of an otitis. It is not improbable, however, that the infective agent may in rare cases pass *directly* into the brain and cause a " primary " cerebral suppuration (Martius). Japha, Lagriffe, Kuchaszewski, and others have lately described cases of obscure etiology.

Brain abscesses due to ear disease are always situated in the corresponding hemisphere. In the great majority of cases they occupy the

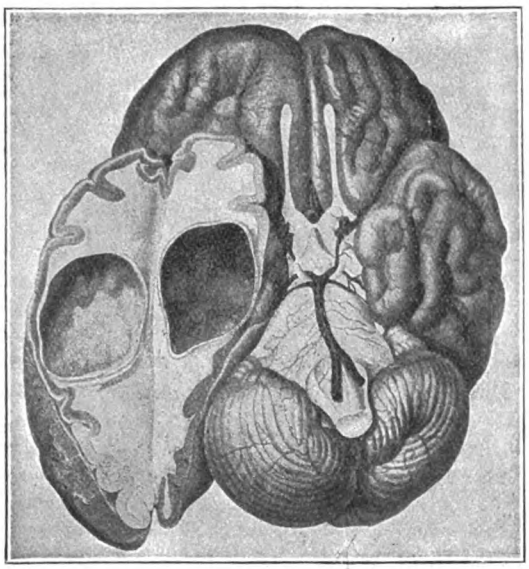

FIG. 322.—Abscess of the right temporal lobe. (After Cruveilhier, reproduced by Ballance.)

temporal lobes (Figs. 321, 322) or the *cerebellar hemisphere* (Figs. 323, 324). Among seventy-six cases collected, the abscess was found fifty-five times in the temporal lobes, thirteen in the cerebellum, four in the cerebrum and cerebellum, twice in the pons, and once in the cerebral peduncle.

In Heimann's recent statistics, there were 456 cases of abscess of the cerebrum to 188 of cerebellar abscess.

It is only in extremely rare cases that a mastoiditis or osteomyelitis of the petrous bone has led, without the intervention of a sinus phlebitis pyæmia, to brain abscesses lying at a distance from the diseased temporal bone, *e.g.* in the optic thalamus in a case by Bauerreis, in the right frontal lobe in left-sided otitis media, in a case described by Japha (see details in Körner, *Nachträge*). Nonne (*Z f. .N.*, xxxiii.) describes an abscess of the frontal lobe which seems to have been due to ear disease. In two other cases (Hegener, Boenninghaus) extensive sinus phlebitis has produced brain abscesses at a distance from the ear. In pyæmia due to otitis and thrombosis, metastatic brain abscesses naturally occur, and these may be situated at some considerable distance from the temporal bone. The failure to recognise this origin frequently leads to abscesses which really arise from suppurative foci in other parts of the body, being attributed to the ear, as Obern-

dörfer has again pointed out (*D. m. W.*, 1906). In one of our cases in which the abscess in a child was on the side opposite to the affected ear, I cannot help suspecting that the examination of the other ear and of the nasal accessory cavities was not a thorough one.

Abscesses in the temporal lobe generally occupy the posterior, inferior segment (third temporal convolution, fusiform gyrus), but they not infrequently extend into or are limited to the region of the occipital lobe. In the cerebellum, the anterior external segment of the hemisphere is usually affected.

Traumatic abscesses are situated in the neighbourhood of the injured part of the skull, specially therefore in the frontal and temporal lobes. *Rhinogenic* abscesses almost always occupy the frontal lobes. The *metastatic* form chiefly affects the vascular area of the arteries of the Sylvian fissure, especially the left.

Traumatic and otitic abscesses are usually *solitary*. More than a single

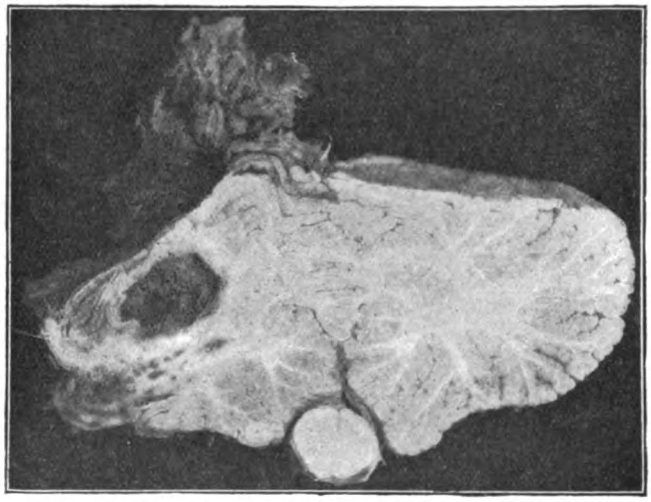

FIG. 323.—Cavity of abscess in the cerebellum, ex otitide. The pus has been evacuated. (Oppenheim.)

focus is only found in about 15 to 20 per cent. of the latter (two close together, or one in the temporal lobe, the other in the cerebellum). Metastatic abscesses are usually *multiple*. Bergmann found over a hundred in a case of pyæmia following gangrene of the leg.

Foci of suppuration in the brain vary greatly in *size*, from that of a pea to that of a fist, of a brain lobe or more. Solitary foci are on an average as large as a walnut or an apple; multiple foci are usually smaller.

R. Müller describes a case of otitic brain abscess, which extended over nearly the whole left cerebral hemisphere, although the pus had twice been evacuated.

The pus is usually green or greenish yellow; it has often a fœtid odour and sometimes contains products of disintegrated brain tissue. It may also have a putrid, ichorous character. R. Müller distinguishes two forms of brain abscess, the parenchymatous and the interstitial. The former have a gangrenous, ichorous character, the latter form true

foci of suppuration, and they alone are encapsulated. He carries this distinction also into the symptomatology.

Long-standing abscesses are, as a rule, enclosed in a solid capsule. This begins to form during the first weeks after the onset of the suppuration, but the membrane only becomes firm in the course of one and a half to two months. This encapsulation of the abscess by no means completes the process; the abscess may extend still further by softening the brain substance or by breaking through the capsule. On the other hand the encapsulated focus may remain for years unchanged in the brain before it passes either spontaneously or on account of some external cause (injury to the head)

FIG. 324.—Site and extent of an otitic cerebellar abscess. Section stained by Pal's method. The lower cavity is an artificial one. (Oppenheim.)

out of this latent stage. The brain tissue round the abscess is usually softened and œdematous. Foci of hæmorrhagic encephalitis may also develop in the more distant neighourhood of the abscess (Oppenheim), or diffuse necrotic processes may take place in the cortex, as in a case of metastatic suppuration described by Dupré and Devaux and attributed by them to toxic influences. Streptococcus pyogenes, staphylococcus pyogenes aureus, albus and citreus are found in the pus of the abscess; in one case it contained pneumococci (Sahli), which are more often found in acute extradural abscesses (Braunstein), and in a few tubercle bacilli. The proteus vulgaris has occasionally been found (Jordan, R. Müller), also micro-organisms identical with or very similar to typhus bacilli, the bacterium coli, and the bacillus pyocyaneus.

Hasslauer gives details regarding the micro-organisms of otitic brain abscesses (*Internat. Zentralbl. f. Ohr.*, v.). With reference to the pathological anatomy of brain abscess and its walls, the recent works of A. Westphal (*A. f. P.*, xxxiii.) and of Kölpin (*Z. f. N.*, xxv.) are worthy of note.

The abscess very often *ruptures into the ventricle*—especially from the temporal lobe—or towards the *meninges*, giving rise to a diffuse suppurative meningitis. *External rupture* towards the nasal cavity, the tympanic cavity, or through the temporal bone is quite uncommon. Amongst the *complications* we would specially mention extradural suppuration, *meningitis*, *thrombophlebitis*, and *sinus thrombosis*. Brain abscess may thus lead to pyæmia, to embolic abscesses in the lungs, and so on. Cerebellar abscesses are very often associated with hydrocephalus.

It is more than doubtful whether a brain abscess may be absorbed spontaneously, although some writers (Ziegler, Macewen) think it possible, at least as regards small foci. The danger is by no means removed by thickening and partial calcification of the contents.

Ballance mentions a case in which the encapsulated brain abscess was so firm and hard that it could be rolled along like a billiard ball (?).

Symptomatology.—The morbid symptoms depend upon the following factors: (1) the suppurative process; (2) the brain disease; (3) the causal disease.

The symptoms are so much influenced by the causal condition that the clinical pictures of traumatic, otitic, and metastatic abscesses differ from each other in many points. But it will be well to describe a morbid picture which includes them all, and then to consider the peculiarities due to the etiology.

In many cases the abscess represents a brain disease of acute onset and acute or subacute course; in others it extends over a longer period, and we may then sometimes recognise three or four stages: (1) the initial stage, (2) the latent stage, (3) that of manifest disease, (4) the terminal stage. The first very rarely comes under observation; the second is usually inferred only from the history of the case, whilst as a rule the third and fourth alone come under medical care, and they may be blended into one.

The description given here therefore refers essentially to the symptoms of this period. With regard to otitic abscesses specially, it should be noted that the initial and latent stages are often absent and unnoticed, because the symptoms cannot be sharply distinguished from those of the ear disease. The affection therefore as a rule immediately reaches its marked or terminal stage and assumes an acute or subacute course—*i.e.* it lasts for some weeks, or less often from one to three months. It is unusual for the disease to have a longer duration, but we should note that even in brain abscess, especially of the metastatic, and less often of the otitic form, remissions and intermissions may occur, even after the onset of the marked symptoms, as in some interesting observations by A. Westphal, Steiner, and others.

In the case described by Westphal, the intensity of the symptoms showed very remarkable temporary fluctuations, the aphasia, paralysis, etc., being sometimes marked, and at other times absent or slight. Even the choked disc improved to some extent, after a small quantity of cerebro-spinal fluid had been drawn off by lumbar puncture.

The symptomatology of *traumatic* purulent encephalitis is practically identical with that of suppurative meningitis of the convexity. Headache

comes on within a few days or one to two weeks after the injury, or it increases in intensity from the time of its occurrence. Vomiting, fever, stupor, general convulsions, excitement, confusion, and less often marked delirium then appear. If the cortical motor zone is affected, the focal symptoms may be associated with those of cortical epilepsy and paralysis of the monoplegic character. The stupor deepens into coma, and death follows within a few days or in two to three weeks ; or there may be a remission, but it is usually only of short duration.

The resemblance to suppurative meningitis at the site of the injury is obvious ; it is only when the symptoms appear after an interval of a week or two, and the course is less violent, showing remissions and intermissions, that we are led to suspect the presence of an abscess in the brain substance. A slow pulse and low temperature are also suggestive, and marked and early onset of the symptoms of cortical foci make it probable that an abscess is the cause of the disease.

Deep-seated brain abscesses, i.e. the late *traumatic*, *otitic*, and other forms, are of much greater clinical interest. In the first named the abscess usually remains *latent* for several weeks, months, or even years (in some cases 10 or 20)—*i.e.* any symptoms to which it gives rise are vague and slight ; the signs of a brain disease then develop acutely or gradually. Careful inquiry and observation show that the latency is often incomplete ; there is headache, which is not considered serious, or a rise of temperature, which is wrongly interpreted, or is attributed to the otitis which may be present. In other cases there are convulsions which are regarded as epileptic or hysterical. There may be emaciation, loss of strength, or mental symptoms, viz., apathy, melancholia, slight confusion, which suggest to the experienced mind a commencing brain disease. These disorders, however, are of less value in making a diagnosis from the fact that in many cases they are inconstant, and that the attacks of brain disease may be separated by intervals of entirely normal health.

The most constant and early symptom of the *manifest* stage of brain abscess of this origin is *headache*, which at times is slight and at others distressingly severe. In many cases it is chiefly felt at a site corresponding to that of the abscess. Percussion of the bone at this point usually produces more or less acute pain. In rare cases it affects chiefly the opposite side of the head. The headache is aggravated by everything which causes congestion of blood or hyperæmia in the head. *Vomiting* is a common symptom, and is very rarely completely absent in cases of cerebellar abscess. The patient usually complains of vertigo.

Fever is a symptom to which great diagnostic importance was formerly attached, but it is rarely high or of long duration in uncomplicated brain abscess, and may be entirely absent in many, indeed in most cases, whilst subnormal temperature is a not unusual symptom.

Okada (" Diagnose und Chirurgie des otogenen Kleinhirnabszesses," Jena, 1900) has carefully studied the temperature in cerebellar abscess. Of 88 cases, 46 showed marked feverish symptoms ; in 15 the temperature was almost normal ; in 15 it was subnormal ; in 8 fever was present only at the commencement, and in 4 it occurred shortly before death. It should be noted that there were often complications in the febrile cases, whilst *as a rule the course of uncomplicated cases was afebrile.* Hoffmann makes similar statements (*D. m. W.*, 1906).

The headache usually becomes worse as the temperature rises. *Rigors* occasionally occur, but they are rarely frequent. *Slowing of the pulse*

is very often noted, and is one of the most important signs; it may be reduced to thirty pulsations. This may be distinctly marked, even when the temperature is high. The breathing may be slow or irregular. R. Müller ascribes the fever and slowness of pulse particularly to "interstitial" abscess (see above). The character and virulence of the micro-organism probably plays a great part in this matter (Ballance).

General convulsions may occur, but they are by no means a constant symptom.

The mental faculties are seldom absolutely unimpaired. There is usually a certain degree of *hebetude*, an inhibition and difficulty of thought, which may at any time become *somnolence*, but the mental powers are as a rule only markedly impaired at the end of the disease. In a few cases restlessness, excitement, and confusion are very marked, or there is violent *delirium*. Apathy and depression are often most prominent. These were specially marked in a case of abscess of the right frontal lobe after influenza described by Dupré and Heitz. The mental condition may undergo change in a short space of time.

A mental disorder of the catatonic type is described by Schäfer, Schmidt (*Z. f. P.*, 1904), and by Kern (*A. f. P.*, Bd. xl.), but the relation of the psychosis to the abscess does not seem to me to be quite beyond question.

Optic neuritis and *choked disc* are present in many cases, but these are much less constant than in brain tumour and are seldom developed to the same degree.

As regards otitic cerebellar abscess, Okada found that out of 46 cases 14 showed optic neuritis on both sides, 6 on the affected side only, and 2 on the opposite side; 3 showed choked disc on both sides, and 4 on the affected side only. The changes were positive, therefore, in more than two-thirds of the cases. Hansen (*A. f. Ohr.*, Bd. liii.) gives the following statistics: Of 12 cases of cerebral abscess, 6 showed a positive ophthalmoscopic change; in 2 there was slight, in 4 marked optic neuritis; of 7 cases of cerebellar abscess, 2 showed optic neuritis. A case reported by Trautmann and Linden proves that choked disc may also appear in rhinogenic abscess of the frontal lobe. Hölscher's remark that he only found choked disc in one of the cases at the Tübingen Clinic is worthy of notice. Lossen (*Beitr. z. kl. Chir.*, Bd. xxxix.) and Sessous (Lucae-Festschrift, 1905) deal with this question.

Takabatake (*Z. f. Ohr.*, 1903) has studied the importance of ophthalmoscopic results as regards the prognosis.

The symptoms often include emaciation, cachexia, and less commonly a jaundiced colouring of the skin. The *gastric* functions are almost always deranged; there is loss of appetite, constipation, bad breath, etc.

Whilst the symptoms just described are due in part simply to the suppuration, and in part to the general affection of the brain and the increase of brain pressure, there are other morbid symptoms which are due to affection of a certain area of the brain, and which must therefore be regarded as *focal symptoms*. These are entirely absent in not a few cases, namely, when the abscess develops in a region of the brain, lesion of which does not produce local symptoms. These include abscesses in the frontal lobes (especially in the right), in the right temporal lobe, and to a certain extent in the cerebellum. It should be noted also that circumscribed foci of suppuration may occupy any site of the brain without causing focal symptoms. I believe, however, that I am right in saying that they are present in the majority of cases, and that they are not always recognised and correctly interpreted.

Abscesses of the *left temporal lobe* are revealed by the very characteristic symptom of word-deafness. I as well as others have been able for this reason to give a local diagnosis in many cases and to indicate the site at which the pus was sought and found. Sensory aphasia was the guide to the diagnosis in the first case of successful operation in otitic brain abscess (Schede). It was formerly often mistaken and regarded as a "mental disturbance." But indeed if the patient is very dazed the diagnosis may be very difficulty. There is seldom complete word-deafness, as in a case of Jordan's, or pure word-deafness (Gehuchten and Goris [1]); much more often there is *partial* or *amnesic* aphasia, *paraphasia*, and, as I [2] have found, *optic* aphasia, the focus of suppuration affecting not the sensory speech centre itself, but the tracts leading to it. The speech affection may be associated with alexia and agraphia.

The occurrence of optic aphasia has been noted, since my observations, by A. Pick and Zaufal (*Prag. med. Woch.*, 1896), Lannois and Jaboulay, Manasse, Brieger, Marie and Sainton, etc., and has been used for definite localisation by A. Pick. I gather from the clinical histories which have been contributed by a number of aurists, that optic aphasia is often overlooked or unrecognised, as the writers proceed on the assumption that in this form of aphasia the object, which cannot be named at sight, would necessarily be at once correctly named if examined by touch, etc. This, however, is not always so, because in examination by palpation the tract from the sensory spheres to the speech centre is not the only one involved, as the visual memory image also plays an essential part. Nor can I agree with Merken's (*Z. f. Chir.*, 1901) interpretation of this affection as a partial mind-blindness. In all his twenty-five cases he found affections of external speech, in one-fourth word-deafness, and in 16 per cent. mind-blindness: Liepmann (*C. f. N.*, 1903), however, to some extent rightly, protests against these statements and their interpretation. I have lately, along with Jansen, seen a very large abscess of the left temporal lobe, in which there was no word-deafness, whilst the visual aphasia was very marked. In a case of Ziehen's, words could not be found even when the memory images for taste were awakened as well as the memory images for sounds. See also Schaffer, *N. C.*, 1907.

Aphasia very rarely occurs in abscess of the right temporal lobe or of the right temporo-occipital region in a right-handed person, as Heine and I have seen in one case ("Verhandl. d. xii. otol. Ges.," Wiesbaden).

Unilateral diseases of the temporal lobe do not produce any affection of hearing (compare p. 631), but Körner draws attention to a case described by Habermann (*Mitt. des Vereins der Ärzte Steiermarks*, 1907), in which an abscess of the right temporal lobe had caused deafness (and anosmia) of the opposite side, which disappeared after a surgical operation. This case is unique.

If the abscess penetrates deep into the white matter of the temporal lobe, it may give rise, either directly or from changes in its neighbourhood, to symptoms which point to a lesion of the *motor*, *optic*, and *sensory* nerve tracts. There is most often a hemiparesis of the opposite side, usually associated with rigidity or contracture. The hemiparesis generally precedes the development of the other signs of pyramidal lesion (exaggeration of the knee-jerks, abnormal plantar-reflex, etc.). Convulsions may occur in the opposite side of the body. Hemianæsthesia and hemianopsia are not uncommon. In a case of abscess of the right temporal lobe observed by Jansen and myself, and operated upon by him, there was hemiparesis and hemihypæsthesia, as well as left bilateral hemianopsia and conjugate deviation of the head and eyes to the right side. In one case which I observed along with Heine, the opposite sensory symptoms were of the character of a pure hemihypalgesia. On the other hand there may be no symptoms of impairment of function of any kind

[1] *Névraxe*, 1903.

[2] *Fortschr. d. Med.*, 1895.

in the opposite side of the body, even in very large abscesses in the temporal lobe, as I have recently seen in a case which was surgically treated by Jansen.

It often occurs that the basal cranial nerves, especially the third and sometimes the sixth, are compressed by an abscess in the temporal lobe, and that this gives rise to corresponding symptoms of paralysis (most often to ptosis).

This fact, which I have previously mentioned, has been again lately observed by Knapp and Sänger (*N. C.*, 1905).

In one such case, it is true, a hæmorrhage was found in the corresponding cerebral peduncle (Kreysig), but this was an unusual condition, as the paresis is due as a rule to lesion of the oculomotor nerve at the base of the brain.

Abscesses of the inferior parietal lobe and of the occipital lobe may cause hemianopsia, which is not uncommon, also, in otitic abscess of the occipito-temporal region.

In a case under the observation of Rotter and myself (*Mitt. aus. d. Grenzgeb.*, vi.), an abscess which occupied the region of the left fusiform and lingual gyri gave rise to no focal symptoms of any kind—to no aphasia or hemianopsia. Even during the probing of the abscess, one could converse with the patient just as if he were a healthy person.

Blindness has been observed in bilateral metastatic abscess of the occipital lobe (Heinersdorf).

It is shown by a case reported by Reverdin and Valette (*Rev. méd. de la Suisse romande*, 1902) that cerebellar symptoms may be produced by an abscess in the occipital lobe.

Abscesses in the motor areas, which are usually of traumatic, but sometimes of metastatic origin—embolic matter very often finding its way from the lungs into the arteries of the Sylvian fissure—give rise to the well-known symptoms of cortical epilepsy and paralysis (monoplegia, hemiplegia). In some cases the cortical paralysis develops gradually under the guise of progressive hemiplegia, and convulsions precede the different attacks of paralysis. This is due to successive stages of suppurative softening of the cortical tissue. Contractures may also develop in the opposite limbs. Large abscesses in the white matter of the cerebral hemisphere usually affect the motor nerve tracts (less often the sensory), so that hemiparesis is one of the most common, but also one of the most indefinite focal symptoms.

Motor aphasia has occasionally been observed in abscesses of the left frontal lobe which are of traumatic, metastatic, or rhinogenic origin.

Severe mental disorders which disappeared with the evacuation of the pus, were observed by Borchard in a case of traumatic abscess of the frontal lobe (*Verhand. d. Kongr. f. Chir.*, 1904).

In cerebellar abscesses the pain is usually felt mainly in the region of the occiput and neck, as a rule on the same side as the abscess and only rarely on the other (as in a case described by Bruns). There is often slight *rigidity of the neck*, and more often still a tendency to hold the head stiff and erect, although this is not due to any real muscular contraction (R. Müller). Focal symptoms may be entirely absent. Severe *vertigo* is to some extent characteristic, and the *affection of co-ordination or cerebellar ataxy* is still more so. This, however, is only very marked in some cases, and has even been absent in abscess of the vermiform pro-

cess, whilst on the other hand it was present in abscesses of other regions of the brain, *e.g.* the temporal lobe, and was very pronounced in one case of rhinogenic abscess of the frontal lobe (Trautmann). Bruns regards it as specially characteristic if the vertigo and vomiting are brought on chiefly by change of the position of the head and body (as in lying on the face in one of his cases), but he rightly states that this also occurs in affections of the labyrinth. In a few cases bilateral amaurosis, due to a complicating hydrocephalus, was a symptom of cerebellar abscess. In any case, optic neuritis and choked disc are comparatively frequent here. Pressure on the pons, corpora quadrigemina, and medulla oblongata, and the cranial nerves arising from them, may also give rise to symptoms such as oculo-motor paralysis, nystagmus, paralysis of conjugate deviation, facial paresis, dysarthria, pain and hypæsthesia in the trigeminal region, and respiratory troubles. In a case which I observed with Jansen I saw diminution of the corneal reflexes and homolateral masseter paralysis, *i.e.* deviation of the lower jaw towards the affected side when it was opened. In a few cases respiratory paralysis appeared, although the heart continued to beat. Interesting cases of this kind are described by Duckworth, Dieulafoy, Hoffer,[1] and Fliess.[2] Spasmodic yawning may also occur. There is occasionally hemiparesis (or in our experience hemiataxia, which is often wrongly regarded as paresis; see also Bruns, *N. C.*, 1904) on the side of the abscess, less often on the opposite side. Lesion of the crossed pyramidal tract may cause spastic phenomena—foot-clonus, Babinski's sign, etc.—on the opposite side (Bruns). On the other hand the knee-jerk was absent in a few cases of cerebellar abscess (Macewen, Müller).

Abscesses of the *pons* and *medulla oblongata*, which are very rare—Cassirer collected ten cases from the literature—give rise, if they are sufficiently large, to the characteristic symptoms of bulbar disease. In a case reported by Eisenlohr in which the abscesses were in the lowest segment of the medulla oblongata, there were no signs of paralysis of the bulbar cranial nerves, but paraplegia was present in all four extremities. The characteristic focal symptoms were present in a case described by Dogliotti, and also in an interesting case of this kind observed by Cassirer and illustrated by Fig. 325. Here the sixth and seventh cranial nerves on the side of the abscess were affected, and neuroparalytic keratitis was present along with hemianæsthesia on the opposite side.

Bregmann gives us an observation of a similar kind (*Z. f. N.*, xxxi.).

Rupture towards the ventricles gives rise to very alarming symptoms: general convulsions, delirium, which rapidly passes into somnolence, tetanic contraction of the muscles of the body, rigor, high fever, etc. The pulse which has been slow, becomes rapid and irregular; breathing is hurried or of the Cheyne-Stokes type. Death occurs within a few hours. In other cases the patient succumbs to the symptoms of a rapidly spreading, universal purulent meningitis.

Diagnosis.—It is often extremely difficult to recognise this disease of the brain. One of the chief grounds of diagnosis is the *etiology*, the detection of some factor which we know by experience may produce brain abscess. When there is no evidence as to its origin, the diagnosis is always absolutely uncertain.

[1] *Prag. med. Woch.*, 1902.

[2] *D. m. W.*, 1903.

If the symptoms of brain disease follow an injury, either directly or after a short interval, there will be no great trouble in distinguishing an abscess from hæmorrhage of the brain or meninges, but the diagnosis as regards suppurative meningitis is a matter of considerable and usually insuperable difficulty. If the symptoms only appear after a long latent stage, we have practically to consider only abscess or brain tumour. The onset of fever, the rigor, the rapid progress of the disease from the time at which it becomes manifest, and absence or late development of optic neuritis are in doubtful cases in favour of abscess. Tumours as a rule have an insidious development, and the symptoms become gradually

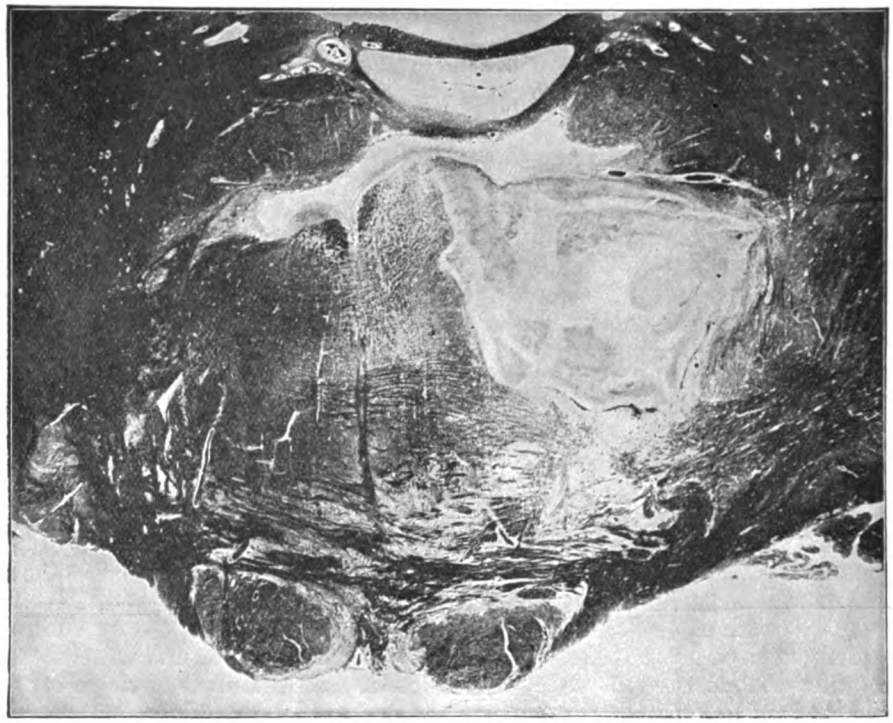

FIG. 325.—Metastatic abscess in the pons and oblongata. (Section by Cassirer in Oppenheim's collection.)

intensified; focal symptoms become sooner evident, and choked disc is almost contantly present, and is often very marked at an early stage. Fever is usually absent.

In a case of traumatic abscess of the occipital region, which I observed along with Lexer, the diagnosis was a matter of greater difficulty, from the fact that the injury had also produced a basal process (fracture, hæmorrhage), and that almost all the focal symptoms were due to this cause.

Traumatic neuroses (see corresponding chapter) may also give rise to confusion with brain abscess of traumatic origin. The diagnosis is not difficult, it is true, when the mental disorders are of the hypochondriacal, melancholic, or hysterical character and the paralytic symptoms are on

the same side of the body as the trauma ; but it is otherwise in the cases in which an injury to the head is followed by vertigo, pressure, stupidity, etc., and in which objective examination reveals no symptoms or merely those of doubtful significance, such as inequality of pupils, rapid pulse, etc. In such cases we should make it a rule to give the diagnosis of brain abscess only on the ground of the more serious symptoms. Fever, slow pulse, and cerebral vomiting on the one hand, and focal symptoms on the other, point in doubtful cases to an organic disease. In contrast to this, functional neuroses are usually revealed by sensory affections of definite character, by vasomotor symptoms, etc. Injuries to the head may also lead to severe symptoms due to fine changes in the vascular system of the brain (Friedmann) and nervous elements, *e.g.* attacks of headache, vertigo, stupor, vasomotor symptoms, etc., but in such cases there are no distinctly focal symptoms corresponding to the site of the trauma, etc.

I have sometimes seen surgical treatment of empyema of the frontal sinuses, followed by the development of marked symptoms of neurasthenia, in which vertigo was as a rule the chief trouble ; but the patients were usually of a neuropathic disposition.

Otitic brain abscess cannot always be immediately recognised, as a number of its symptoms also occur, especially in youth, in uncomplicated otitis, and in particular during exacerbations of the chronic process and retention of pus. Amongst these are headache, vomiting, vertigo, stupor, fever, and even inequality of pupils and diplopia, especially paralysis of the abducens (Körner, Peyser, Alt, Terson). It would seem from a few cases that even optic neuritis and ptosis may be due to this cause. All these symptoms have disappeared, for instance, after paracentesis of the tympanic membrane, or after removal of a granular polypus (Roughton). When therefore they occur in the course of an ear affection, we should first suspect a local cause, such as retention of pus. If they persist or return after evacuation of the pus and suitable treatment of the ear disease, and if they cannot be explained by the local process, then the diagnosis of brain abscess becomes more certain (if serous meningitis can be excluded). The appearance of optic neuritis in affections limited to the ear is so unusual that its presence must suggest an intracerebral disease.

The symptoms of *labyrinth disease* and of *Ménière's vertigo* should very rarely give rise to confusion, but they have in a few cases simulated the picture of cerebellar abscess. The combination of headache with vertigo, vomiting, and fever, and even with nystagmus, may be due to a simple disease of the labyrinth.

The differential diagnosis of cerebellar abscess and suppuration of the labyrinth has recently been thoroughly discussed by Neumann (*A. f. Ohr.*, Bd. lxvii.). See also Hinsberg (*D. m. W.*, 1906).

External purulent pachymeningitis or *extradural* (epidural) *abscess*, the most frequent complication of suppuration of the ear, may, during life, give rise to almost all the symptoms of brain abscess, but the symptoms of cerebral pressure are as a rule much less marked, and the focal symptoms are much more often absent ; hemianopsia is hardly ever present. But the most important differential signs are the local symptoms of this affection, the swelling and intense painfulness of the region behind the mastoid process, the presence in many cases of a fistula, the limitation of the movements of the head, torticollis, etc. I have also in two cases

found marked dulness of the percussion note on the corresponding area of the skull. In other respects the differential diagnosis is not of great practical importance, as when in doubt we should always look first for the extradural focus of suppuration, which will be revealed by any radical operation directed to the disease of the ear.

For further details regarding extradural and intradural abscess (pachymeningitis interlamellaris and intrameningeal or subdural abscess) consult Körner. Reviews on the subject of extradural otogenic abscesses are also published by Braunstein (*A. f. Ohr.*, Bd. lv.), Hölscher ("Die otog. Erkr. d. Hirnhäute," etc., Halle), Heine ("Festschrift Lucae," 1905).

Otitic sinus thrombosis is as a rule combined with high, remittent fever, the temperature rising and falling abruptly, rigors, diffuse perspiration, pyæmic symptoms, metastases, etc., and with the *external* symptoms of sinus thrombosis, especially those arising from the extension of the process to the jugular vein (compare the following chapter). There are, however, atypical cases in which the clinical picture resembles that of abscess and *vice versâ*.

The greatest difficulty attends the diagnosis of abscess from *diffuse purulent meningitis*. In typical cases, it is true, the symptomatology of this disease differs essentially from that of abscess, by its acute onset, rapid course, high fever, rapid pulse (slowing of the pulse is seldom of long duration and never very marked in degree), and symptoms of meningeal irritation ; by cutaneous and sensory hyperæsthesia, by mental and physical unrest, convulsions and transient tremors, muscular rigidity, spinal symptoms, etc. etc. There are other cases, however, quite atypical in their course, which show a clinical picture entirely resembling that of abscess. It may ultimately be impossible to determine from the symptoms whether the abscess is associated with meningitis or not. In lumbar puncture, however (see p. 762), we have a method which usually leads to a definite conclusion (Lichtheim, Leutert), as in brain abscess the fluid is almost always clear and free from organisms, whilst in generalised purulent meningitis, if the communication between the cavity of the skull and the spinal canal is open, the fluid is turbid, rich in cells, and contains bacteria. An ever-increasing number of anomalous results, however, such as those of Lichtheim, Stadelmann, Brieger, Reverdin-Vallette, Passow-Voss, Wolff, Rupprecht, Neumann, and Gerber, show the necessity for caution in this matter. According to Körner slight turbidity and presence of bacteria in the fluid do not indicate the existence of diffuse meningitis. Some aurists would resort to lumbar puncture in every case for the purpose of diagnosis, regarding it as free from danger, whilst others (Lucae-Heine) would use it only when the interests of the differential diagnosis absolutely demand it. Körner takes this latter point of view, and he agrees with us in regarding the procedure as not without risk.

He does not, it is true, regard the results of lumbar puncture as absolute proof, but he thinks that the presence of pus and bacteria in the fluid is, as a rule, evidence of the pre-existence of an already fully developed *purulent* lepto-meningitis. This method was employed in Passow's Clinic, as Voss tells us (*D. m. W.*, 1903), in every case in which there was any suspicion of an intracranial complication, but even there some serious collateral effects, such as hæmorrhage, were observed. Grunert (*M. m. W.*, 1905) insists upon its value, although he recognises its danger. Frey (*W. m. Pr.*, 1905) states that the difficulty of diagnosing between abscess and meningitis is not overcome even by lumbar puncture, and this is shown by the observations of Gerber (*D. m. W.*, 1906), Hoffmann (*B. k. W.*, 1906), and others.

As regards the value of brain puncture in diagnosis, see further on.

In doubtful cases a *slow pulse*, a slightly abnormal temperature, simple increasing stupor, and all the symptoms which point to a circumscribed localisaton of the lesion (in the temporal lobes or cerebellum), are evidence in favour of brain abscess.

Localised purulent meningoencephalitis, in some cases, especially when it is situated above the temporal lobe or cerebellum, produces a clinical picture exactly like that of brain abscess. Such interesting cases of subdural abscess have been recently described by F. Krause (and Jansen) and by Heine. We should also refer to this chapter regarding diagnosis of abscess from *meningeal tuberculosis*. Hinsberg has lately described two cases in which the picture simulated that of brain abscess.

Serous meningitis, a frequent complication of purulent otitis, may on the one hand simulate the symptoms of brain abscess, on the other that of purulent meningitis. But as a rule it does not give rise to marked focal symptoms (bilateral paresis of the extremities, unilateral or bilateral paralysis of the cranial nerves, and cerebellar ataxia have been more often noted) or to severe and persistent pyrexia, whilst choked disc and marked affection of the sight is a common and early symptom. Moreover it is specially important to note that it may show spontaneous improvement, or may disappear after the removal of pus which has been retained in the ear or in the extradural region, and especially after surgical operations which are associated with opening of the subarachnoidal space. Cases of this kind have been recorded by Macewen, H. Levi, Kretschmann, Beck, Waldvogel, Müller, Lecené-Bourgeois, Broca-Laurens, and others, and I, along with Bergmann and Jansen, have seen a severe case of this kind which recovered after lumbar puncture.

I have been able of late years to observe several cases of this kind. In one there were symptoms arising from the abducens, oculomotor, and vagus, so that our diagnosis inclined rather to be one of a circumscribed basal suppuration. These all disappeared with the withdrawal of large quantities of fluid. It is not clear whether the case was one of compression of the basal cranial nerves from the œdematous brain (Reichardt's acute brain swelling) or of a simultaneous neuritis. Serous meningitis may also lead to unilateral labyrinthine symptoms (Oppenheim).

Lucae in one case noted a profuse flow of cerebro-spinal fluid which persisted for five weeks. Lumbar puncture is certainly a valuable aid in the diagnosis of this disease, but it does not enable us to distinguish with absolute certainty between abscess, serous meningitis, and sinus thrombosis. The frequent occurrence of this affection in diseases of the ear explains the fact that in many cases (Gradenigo, Röpke, R. Müller, etc.) in which an operation was performed for a supposed but non-existent brain abscess, the symptoms of the grave brain disease completely disappeared after the operation.

The cases of Herzfeld (*B. k. W.*, 1905) and E. Meyer show that the disease occurs in the corresponding processes of the nose and its accessory sinuses, and may lead to diagnostic errors. Traumatic serous meningitis may also suggest a diagnosis of abscess; thus in one case of gunshot penetrating into the wall of the lateral ventricle, I noted convulsions, followed by slowing of the pulse, optic neuritis, and disappearance of the tendon reflexes. There was no pyrexia. I diagnosed serous meningitis, and spontaneous recovery took place.

Acute non-purulent encephalitis may—as I have pointed out and observed in a number of cases—accompany a disease of the ear, but here the characteristic topographical relations to the ear affection are absent,

and the diagnosis can also be made from its development and course. It is true that when the lesion is on the left side aphasia is a common symptom, but as a rule it is not sensory but motor aphasia, and its incomplete forms, as this affection, much more often involves the frontal lobe and central area than the temporal lobe. But we should not forget that it may be localised in the temporal lobes, as I have lately seen (compare the chapter on encephalitis).

A few aurists (Merkens, Müller) think that local œdema or "serous encephalitis" may result from otitis and give rise to focal symptoms.

Confusion with *cerebral tumour* has occasionally arisen (Passow, Schwartze), but I have always been able to avoid this, even where the conditions were favourable for development of an abscess (compare chapter on brain tumour).

Nonne (*Z. f. N.*, xxxiii.) has lately described two cases in which the abscess completely simulated the picture of brain tumour; these were really due to a tubercle.

Hessler has collected nineteen cases of brain tumour in disease of the ear, two-thirds of which had not been diagnosed (*A. f. Ohr.*, Bd. xlviii.).

I must not fail to mention a *reflex neurosis* which I have several times noted in disease of the ear. Whilst the subjective symptoms often greatly resembled those of abscess, there were in almost every case signs of paresis and anæsthesia with diminution of the sensory functions *on the same side of the body as the affected ear*, and these afford a sure foundation for the diagnosis.

A girl whom I treated for this disease localised all her symptoms in the side of the body corresponding to the affected ear. Even in her dreams these occupied her mind to such a degree that she scratched the skin off the limbs on this side during sleep.

The *local diagnosis* of otitic abscess, especially the determination as to whether it is situated in the temporal lobe or the cerebellum, is by no means always a matter of certainty. Thorough examination of the ear, the site of the carious process, the localisation of the pain and sensibility to percussion, furnish us with certain conclusions, but the above-mentioned focal symptoms are those which determine the localisation. We may accept it as an axiom that when only one ear is affected, the abscess will be found on the corresponding side. Exceptions to this rule hardly ever occur except in pyæmia (see above), and here the abscess is not an otitic, but a metastatic one.

Abscess of the temporal lobe in general, especially when on the left side, can be diagnosed with much greater certainty than abscess of the cerebellum. If we consider the cases of cerebellar abscess in which an operation has been successfully performed—Koch mentions nineteen, but the number has been greatly increased since then—we find that the local diagnosis has not usually been indicated by the nervous symptoms, but by the otological condition (necrosis of the anterior wall of the posterior cranial fossa, dural fistula, etc.), or that the cerebellum has only been explored after the pus has been sought in vain in the temporal lobe. Okada was able to collect seventeen cases in which the disease had been unrecognised. The following facts should be considered in diagnosis: Evidence in favour of a cerebellar localisation of the focus of suppuration, apart from the ear condition, is afforded by cerebellar ataxia, if it is severe and develops early, by the localisation of the pain and cutaneous

sensibility in the occipital region, rigidity of the neck or a stiff attitude of the head, well-marked nystagmus,[1] choked disc with grave affection of sight, symptoms arising from the pons and medulla oblongata, etc. etc. Patients suffering from cerebellar abscess are usually inclined to lie upon the unaffected side (Gradenigo, but this is opposed by the cases of Raughton-Cunning, etc.). Hardly one of these criteria is in itself absolutely trustworthy, but taken together they are exceedingly valuable. On the other hand we should also consider the negative signs, such as absence of aphasia (when the focus is on the left side), of hemianopsia, etc. Homolateral paresis (?), and specially homolateral ataxia of the extremities, has often been noted in cerebellar foci, but not in foci of the temporal lobe. On the other hand spastic paresis of the opposite side may occur in cerebellar abscess also. The corresponding symptoms are important in so far that they differentiate abscess from affections of the labyrinth, but they are of little value as regards special localisation. Partial paresis of the oculo-motor nerve, ptosis in particular (Oppenheim, Wilbrand, and Saenger), is more frequent in abscesses of the temporal lobe. Okada states that in abscesses of the cerebellum the mind remains unaffected until the last stage.

There may be no focal symptom whatever in rhinogenic abscess of the frontal lobe, as in Herzfeld's case.

I am sceptical as regards the statement that X-ray examination may afford positive grounds for the diagnosis (Sträter, " Forschr. d. Röntgen," vii.).

Brain puncture, as used specially by Neisser and Pollak, is an aid to diagnosis which should not be undervalued. Although objections have been raised to the use of this method in the diagnosis of brain abscess (Borchardt, F. Krause), it seems to us that we are justified in resorting to it in doubtful cases, more especially in order to establish the local diagnosis. The danger of meningeal infection cannot be very great, as it is almost always immediately followed by a radical operation, sometimes with positive results.

A spot situated upon a line drawn vertically from the superior insertion of the auricle, some 0·5 to 0·75 cm. above this insertion (see Fig. 326), may be regarded as the most common site for abscess of the temporal lobe. The second temporal gyrus is then usually affected in its basal parts. In cerebellar abscesses the part lying in the sigmoid fossa is as a rule affected. It is most successfully localised between two points, one of which is in the middle of a line from the inion to the tip of the mastoid process, whilst the other corresponds to the posterior superior angle of the mastoid process, *i.e.* to the highest palpable point of the posterior margin of the mastoid process.

Details as to the technique and the avoidance of hæmorrhage may be found in the original work of Neisser-Pollak (*Mitt. aus d. Grenzgeb.*, xiii.) and in the *Verhandl. der Gesellsch. Deutcher Nervenärzte*, 1907.

Prognosis.—Brain abscess is a disease which almost always ends

[1] Lange (*D. m. W.*, 1907) thinks that the existence of nystagmus towards the side of the affected ear is in favour of cerebellar abscess, but Wagner has seen it in abscess of the temporal lobe. Moreover, the ear disease or affection of the labyrinth may in itself give rise to nystagmus, although it is then as a rule directed towards the unaffected side. As regards the attempts to distinguish between laybrinthine and cerebellar nystagmus, see Neumann ("Der otit. Klinhirnabszess," Wien, 1907, and *A. f. Ohr.*, 1906), Barány (*loc. cit.*), etc

fatally if surgical aid is not sought. It may indeed rupture into the tympanic, buccal, or nasal cavities, or to the outside, but on the whole this is a very rare occurrence, and spontaneous recovery hardly ever takes place in this way. Such a rupture is more common in extradural abscess (Braunstein). Ziegler believes, as already mentioned, that small abscesses are capable of spontaneous recovery, but this is hardly of any practical

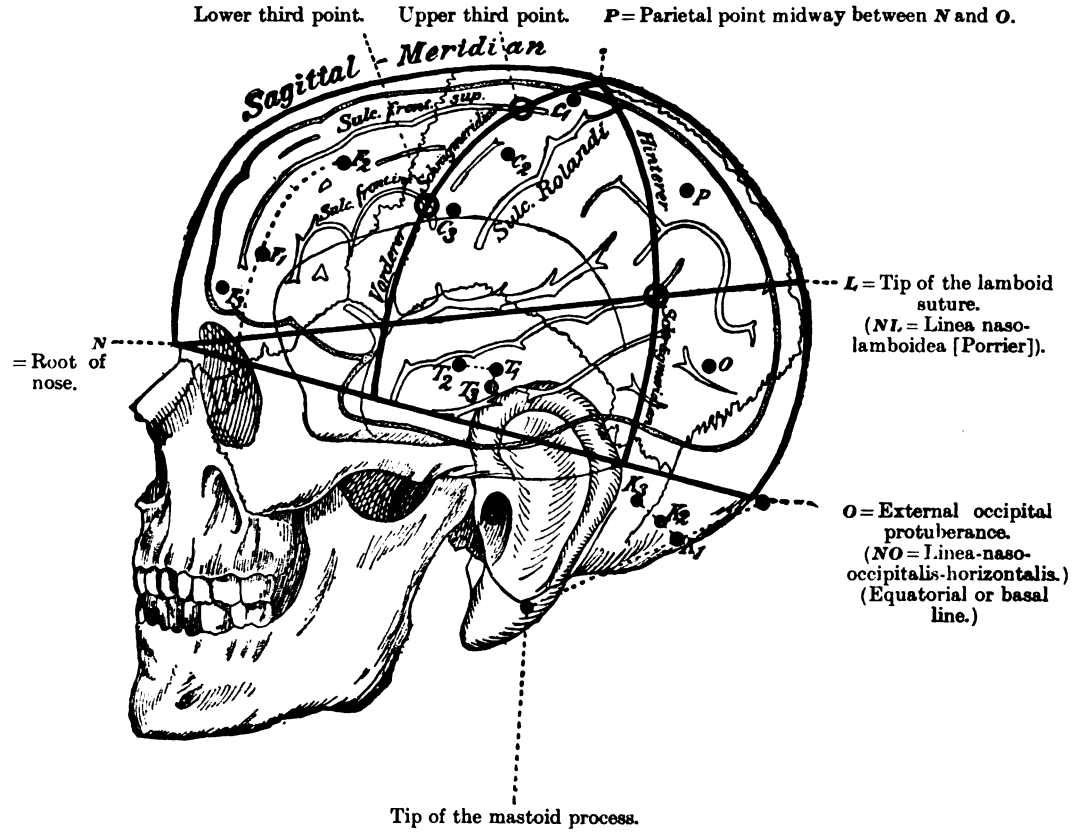

FIG. 326.—Diagram as a guide to cranio-cerebral topography. (After Poirier-Kocher.) Drawing of Neisser's points for trephining.

— Kocher's craniometer points or lines. ◍ Neisser's abscess points.

● Neisser's points for trephining of the various lobes.

Vorderer Schrägmeridian = Anterior oblique meridian.
Hinterer Schrägmeridian = Posterior oblique meridian.

importance. In the great majority of cases, therefore, death ensues, either from the development of œdema of the brain and hydrocephalus, or from bursting of the abscess into the meninges or ventricles. The patient usually dies in deep coma. The patient's fate is sealed as soon as the disease enters on its final stage, if the abscess is left to itself.

The prognosis has, however, somewhat improved since we have succeeded in curing traumatic late-abscesses and otitic abscesses by surgical intervention.

Out of 60 cases of brain abscess of traumatic origin in which opera-

tion was performed, 38 were cured, and out of 138 of otitic abscess, 62 recovered. These statistics, which were given in the third edition of this text-book, have been since then supplemented by such a great number of others that they can hardly be brought under review. From a superficial perusal of the literature at my disposal I find that from the years 1898 to 1901 (July), there were reported over 196 cases of otitic brain abscess with 34 recoveries, so that altogether we have 96 cures in 196 cases—almost 50 per cent. These statistics are similar to those given by Körner, who reports 72 recoveries out of 140 cases, *i.e.* 51·4 per cent. Körner's later statistics taken from the year 1901, comprise 212 operations for cerebral abscess, with 52·8 per cent. of recoveries, and 55 of operations for cerebellar abscess, with 52·8 per cent. recoveries (for all particulars see Körner's masterly work). But in how many cases have unsuccessful operations remained unpublished! Our conclusions from such statistics must therefore be drawn with great reserve. Körner himself has repeatedly emphasised this fact. The results obtained vary greatly in different operations. Macewen has been the most successful, as having eighteen recoveries out of 19 cases of brain abscess upon which he operated, and in one of these cases which recovered the abscess was complicated with purulent meningitis. Körner in his latest publication summarises the results reported from several clinics as follows: Of 23 cases in which the cerebral abscess was found and evacuated by operation, 11 recovered, whilst 12 died; of 15 cases of cerebellar abscess only 4 were cured. There are others in which the abscess was not found, so that the result is that of 30 cases of brain abscess from the clinics of Schmiegelow and Körner, only 8, or 26·6 per cent. were cured.

We may also allude to the less extensive statistics of such writers as Uchermann (*Norsk Mag. f. Lägar*, 1904), Voss, Bibrowicz (*Beitr. z. klin. Chir.*, Bd. xlvii.).

The permanence of the recovery was confirmed by Saenger in one case after twelve years, and in one of my own after ten years.

In many cases it was necessary to evacuate the pus several times. In a case of Whitehead's [1] there was an interval of two and a half years between the first and second operations, but the intervals are usually shorter. In Gluck's [2] well-known case the operation induced recovery which lasted for eleven years; then headache and epilepsy supervened, and a cyst was found at the site of the previous abscess. When this was evacuated and excised, recovery again followed. Ledderhose saw an absolutely similar course in a traumatic frontal abscess.

The fact that the operation is in a number of cases followed in a short time by death should not preclude operation in a disease which is in itself fatal. On the other hand it should not be forgotten that operations are sometimes performed under a wrong diagnosis, and that in this way the life of a patient suffering from a disease capable of spontaneous recovery may be endangered. I have known two cases of this kind in which the operation was certainly responsible for death, and a third in which a patient suffering from a neurosis was operated upon under the diagnosis of abscess, with the result that he became aphasic. I should state that I only knew these patients after the operation. In a few cases with a favourable course (such as a case of Heine's which I examined, and another observed by Waterhouse) the aphasia only developed after the evacuation

[1] *Lancet*, 1904. [2] *D. m. W.*, 1905 and 1906.

of the pus, or has only been diagnosed since the mental condition cleared up. Under these conditions, however, it always disappears, and in Heine's case it did so after a couple of days.

Optic neuritis or choked disc may also first appear after evacuation of the pus, as in Sachsalber's case (*Z. f. Aug.*, ix.).

Abscesses of the temporal lobe afford on the whole a much better chance for surgical intervention than those of the cerebellum. The less favourable prognosis of the latter is specially shown by the statistics of Okada and Körner.

B. Manasse (*D. m. W.*, 1904), for instance, was able to show to the Strassburg Medical Society three cases of recovered cerebellar abscess.

So far there have been only a few cases of successful operation upon rhinogenic abscesses of the frontal lobe (Denker, Herzfeld,[1] Zalewski, Hamersfahr [2]). We have no information as to the ultimate result of cases, such as that described by Rafin, which have undergone repeated operation.

The prognosis as regards surgical treatment of uncomplicated extradural abscesses is on the whole favourable. Thus Denker (*Z. f. Ohr.*, Bd. xliii.) was successful in all the five cases upon which he operated.

Treatment.—The most important point of all is *prophylaxis*. The onset of brain abscess is to be prevented by protecting wounds on the skull from infection, by rendering foci of infection in the neighbourhood of the brain innocuous, and by guarding the brain from infection. Details as to the measures which should be adopted for this purpose, *e.g.* the treatment of injuries of the cranial bones, of otitis, of retention of pus in the wound and in the spaces of the temporal bone, etc., will be found in the text-books on otology and surgery.

The increasing rarity of otitic brain abscess is noted, *e.g.* by Takabatake (*Z. f. Ohr.*, 1903), and is attributed to these factors. It is my impression also that brain abscess has become much less common of late years.

Bergmann cautions us specially against forced syringing and irrigation of the ear.

Evacuation of the pus is indicated in every case where the diagnosis of traumatic brain abscess can be definitely established and an incurable complication excluded. A. Bergmann would even apply surgical treatment to red softening of traumatic origin.

Surgical treatment is suitable for every case in which there is a certain diagnosis of uncomplicated brain abscess, and in which there are no evident symptoms of rupture or of any other disease which would in itself prove fatal. It is advisable as a rule to adopt the method which first deals with the affection of the ear and subsequently with that of the brain, in order to ascertain whether the relief of the pus retained in the ear or in the extradural region will cure the brain symptoms. Where this does not follow, or where the result is negative, the abscess in the brain should be found and evacuated.

In a few cases, however, where the local and general diagnosis leaves

[1] *B. k. W.*, 1901. [2] *D. m. W.*, 1907.

no room for doubt, this rule should be disregarded and the abscess in the brain should be directly attacked.

For details as to technique, consult the work of Bergmann, Macewen, Körner, and also Jansen, Schmiegelow (*Hospitalstid.*, 1904), Heine (" Die Operationen am Ohr," etc., Berlin, 1904), etc.

External purulent pachymeningitis, sinus thrombosis, and even commencing pyæmia afford no contraindication for surgical treatment, nor does circumscribed purulent meningitis.

If a diffuse purulent meningitis is present, it would seem advisable to abstain from operation. Some writers, however, encouraged by the results of Macewen, Beck, Gradenigo, Lucae, Gerber, Sokolowsky, and others, have even, under such circumstances, advocated trephining. Barbarin also takes this point of view. From the experience of recent years I must also say that I do not regard operation for abscess as contraindicated by the suspicion of a general purulent meningitis. In this opinion I rely mainly on the fact that symptoms of a diffuse purulent meningitis may occur in the initial, or even in the latent, stage of abscess (Körner). Lumbar puncture may decide the question as to whether the brain abscess is associated with diffuse purulent meningitis, but this indication can by no means be entirely relied upon (see above). Lumbar puncture should be performed with Krönig's instrument, and every precaution should be taken. Sudden and material diminution of the pressure should be avoided with special care.

Surgical treatment is unsuitable for metastatic abscesses, as they are almost always multiple and due to a primary and often incurable disease. Two cases upon which Ziehen [1] operated ended fatally; in one of these the diagnosis had been at fault. Cure has, however, been effected in a few cases of this kind.

Surgical treatment has occasionally been applied to typhoid abscess (Keen, Cassels-Brown).

Thrombosis of the Cerebral Sinuses

Literature: Gerhardt, " Deutsche Klinik," 1875; Nothnagel, " Ziemssen's Handbuch," xi.; Leichtenstern, *D. m. W.*, 1880; Zaufal, *A. f. Ohr.*, xvii.; Bollinger, *M. m. W.*, 1887; Wernicke, *Lehrb. d. Gehirnk.*, iii., 1883; Horsley, *Br.*, 1888; Jansen, *A. f. Ohr.*, xxxv.; Jansen, *Volkm. Samml. N. F.*, 1895; Macewen; Körner, *loc. cit.*, Bücklers, *A. f. P.*, xxv.; Zaufal, *Prag. med. W.*, 1897; Hölscher, *Die otit. Sinusthrombose*, 1902; Goldschmidt, " Die otit. Pyämie, Sammelref.," *C. f. Gr.*, 1903; Leutert, *A. f. Ohr.*, Bd. lxi.; Halff, " Die marant. Thromb.," Dissert., Basel, 1904; Kümmel, " Die v. Ohr ausgehenden sept. Allgemeininf. Mitteil. aus d. Grenzgeb.," 1907; Brieger, *B. k. W.*, 1907.

Comprehensive digests are also given by Uhthoff, " Graefe-Saemischs Handbuch," 2nd ed., T. xi., and *M. f. P.*, xxii.

Anatomy (according to Leube)

The transverse sinuses, which pass through the transverse grooves, open, along with the inferior petrosal sinus, into the internal jugular vein (see Fig. 327 and Fig. 253, p. 607). In addition to the sinuses which open into the Torcular Herophili, the cerebral veins and the external veins of the skull also convey blood into the transverse sinus; the occipital veins and the posterior auricular, which unite before entering the external jugular vein, by means of a branch passing through the mastoid foramen, and the veins of the neck by means of a branch which passes through the posterior condyloid foramen and forms the connection between the transverse sinus and the plexus of veins in the neck.

The superior longitudinal sinus runs from the foramen cœcum of the frontal bone, by which

[1] Ebstein-Schwalbe's " Handbuch d. prakt. Med.," 1900.

it is connected with the internal veins of the nose, to the internal occipital protuberance. It is connected with the external veins of the skull by emissary veins as well as by its tributaries from the brain (superior cerebral veins) and from the falx cerebri. The cavernous sinus, at its anterior end, receives the superior ophthalmic veins, the upper branch of which is connected with the anterior facial vein at the inner canthus (Figs. 328, 253).

For particulars as to the occipital sinus, its connections with the transverse sinus and its varieties, see Henrici-Kikuchi, *Z. f. Ohr.*, Bd. xlii., and Körner, Supplement to the 3rd edition.

Sinus thrombosis is seldom a pure, uncomplicated disease. A clear outline of its symptoms can hardly be given, as in the great majority of cases they have to be selected from a clinical picture produced by a combination of morbid conditions.

One may speak of a *primary* and of a *secondary* sinus thrombosis. The latter is due to *phlebitis* of the wall of the sinus or of one of the veins opening into it, and this phlebitis is again almost always the result of

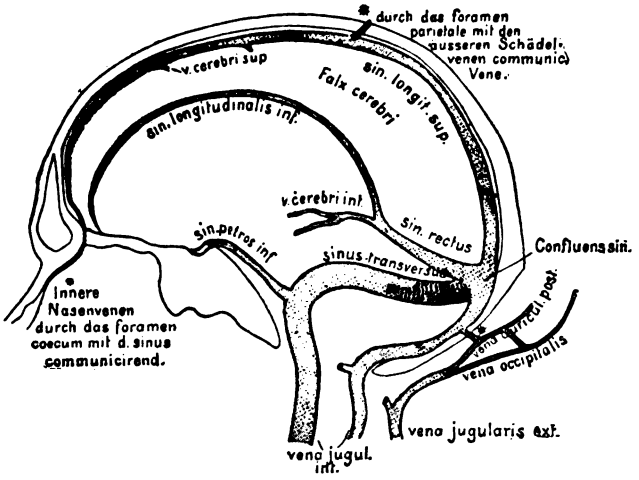

FIG. 327.—(After Leube.) Diagram of the connections of the superior longitudinal and transverse sinuses with the external veins.

* Communication through the parietal foramen with external veins of the skull.
* Internal nasal veins communicating through the foramen cœcum with the superior longitudinal sinus.

an *inflammatory* (usually septic) *process in the neighbourhood.* Secondary thrombosis is therefore usually of an infective nature.

With regard to the causes of sinus thrombosis the following facts are known to us:—1. Primary thrombosis is due as a rule to *weakness of the heart*, and is therefore known as *marantic.* It occurs chiefly in children under the age of one year and in old people. In children it is mainly caused by exhausting diarrhœa. The weakness of the heart causes slow circulation of the blood. The conditions of the circulation in the sinuses tend to the recurrence of coagulation within them.

Coagulation is also aided by the blood becoming thickened and diminished in quantity, especially as the sinus wall, being inelastic, does not accommodate itself to the amount of blood within it. The trabeculæ which pass through the sinus also help to induce coagulation.

The disease may develop in the course or at the termination of some

exhausting illness, e.g. pulmonary consumption, carcinoma, etc. It occurs less frequently in the course of acute infective diseases, such as typhoid, pneumonia, or smallpox. It may apparently be due to nephritis (Voss). The fact, first demonstrated by Bollinger, that *chlorosis* is a not uncommon cause of sinus thrombosis, is of special importance. Cases of this kind have been reported by Andrew, Church, Proby,[1] Pasteur,[2] Göbel,[3] Nonne, Kockel,[4] Schweitzer, Hawthorne,[5] Lewis, and others. In addition to the heart weakness, which is a factor here also, *fatty degeneration of the endothelium of the sinus*, which deprives the trabecula of its lining, and increase in the blood plates tend to promote coagulation.

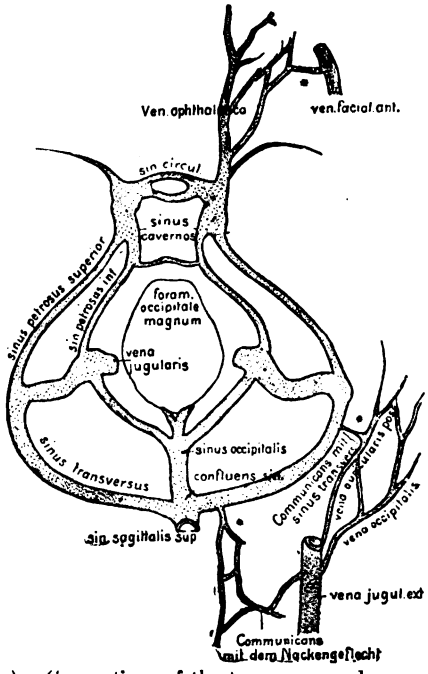

FIG. 328 (after Leube).—Connections of the transverse and cavernous sinuses with the external veins (*).

Simple anæmia, on the other hand, is very seldom the cause of thrombosis of the cerebral sinuses; but it has been observed after repeated hæmorrhages.

A few cases of autochthonous sinus-thrombosis have been observed, for which no cause could be ascertained. The cases described by Good and by Walko (*Z. f. Heilk.*, 1903) are also of unknown origin. (In the latter of these the thrombosis followed surgical removal of a uterine polypus, and arose possibly from the paravaginal veins.)

2. *Secondary* phlebitic sinus-thrombosis is usually due to affection of the bone adjoining the sinus or connected with it by means of veins. *Otitic* thromboses are much the most important of all the forms which have this origin. Otitis media, caries of the petrous bone, and cholesteatoma are very common causes of sinus thrombosis. According to

[1] *Thèse de Lyon*, 1889.
[2] *Lancet*, 1888.
[3] Dissert., München, 1893.
[4] *A. f. kl. M.*, 1894.
[5] *Brit. Med. Journ.*, 1902.

recent observations, the primary affection in the temporal bone is more often acute than chronic (Jansen, Voss, Körner). Thrombosis may also develop after acute otitis. The carious process is transmitted directly to the sinus wall, or the *thrombo-phlebitis* of a vein which connects the inner ear with the sinus extends to the sinus itself. In such cases there is very often a complication with *meningitis*, or with *extradural or brain abscess*. There is also as a rule *pyæmia*, due to the septic character of the thrombosis, the septic matter finding its way into the jugular vein and thence into the circulation.

Caries of other bones of the skull, purulent and tubercular meningitis, and suppurative, septic diseases on the skull, in the face and its cavities (orbit, nasal cavities, possibly also the antrum of Highmore), may also give rise, by means of the parietal emissary vein, to thrombosis of the sinuses, for almost every sinus receives veins from the exterior of the skull. By means of these, injuries, carbuncles in the face or neck, parotitis, facial erysipelas, orbital phlegmon, panophthalmitis, etc., may produce sinus thrombosis (dental caries has even been supposed to be a cause in some cases). It may also be merely one of the symptoms of general septicæmia or pyæmia.

The *tubercular* form of sinus phlebitis is a rare disease. Hartmann, Piffl, and Grossmann (*B. k. W.*, 1904) have described cases of this kind.

Nestor (*Spitalul*, 1905) thought that staphylococcus-thrombo-phlebitis of the cavernous sinus in one case was due to a hordeolum.

Finally, the disease may be of *traumatic* origin and may be due to compression of the sinus, *e.g.* by tumours.

In one case (Dench), the sinus thrombosis was caused by injury of the bulb of the jugular vein in extraction of the auditory ossicles. Severe concussion from a fall may have the same effect, as the experiments of Dörr have shown to be probable.

In two cases of apparently primary (spontaneous) thrombosis of the sinuses, Huebner found micro-organisms in the thrombus and in the cerebro-spinal fluid.

Pathological Anatomy.—The thrombosis may be confined to one sinus and to one part of it, or it may extend to several, and even to all the sinuses. Thus Bückler describes in one case thrombosis of almost all the sinuses and veins of the brain, and Patel has seen a similar one. The affected sinus is occupied by a fresh, grey-red thrombus or an older firm, pale one which is adherent to the wall. The older the thrombus, the more firmly is it attached to the wall. A secondary thrombus is usually discoloured, puriform and sanious, and in such cases the wall of the sinus has a peculiar appearance; it is green or greenish yellow in colour, etc. Purulent matter is found not only in the sinus itself, but also in the jugular, subclavian, and even in the vena cava superior. The work of Körner should be consulted for information as to the micro-organisms found in sinus thrombosis. These include streptococci, the proteus vulgaris, the bacillus pyocyaneus, etc. In cases of phlebitic thrombosis, abscesses are often found lying beside the wall of the sinus. The thrombi usually extend into the veins which open into the sinus. Thus in thrombosis of the great longitudinal sinus, the veins at the convexity of the brain are often transformed into firm columns of a purplish colour, "into black, tense tubes." The thrombosis may extend from the straight sinus into the vena magna Galeni and the

internal cerebral veins and may give rise to hydrocephalus. (This is by no means, however, a constant or even a common result of sinus thrombosis, as some writers think.) The part of the vein which is not thrombosed is very tortuous, dilated, and distended with blood. The areas of the brain from which these veins derive their blood supply are hyperæmic and permeated by capillary, sometimes by large hæmorrhages; infarcts and foci of softening are very often, and abscesses occasionally found. Meningeal hæmorrhage is less frequent. The complications of secondary thrombosis have already been described. The heart is said to be abnormally small in a surprising number of marantic cases.

The *site* of the thrombosis varies according to its *cause*. The marantic form is almost always localised in the longitudinal sinuses, especially in their posterior portion, and it may involve the transverse sinuses. Otitic thrombosis usually involves the transverse sinus (in the sigmoid fossa), sometimes the cavernous, the superior or inferior petrosal sinus, sometimes several sinuses at the same time. It may extend into the longitudinal sinus, and is not infrequently propagated from one sinus into another, even into those of the opposite side of the brain. An extradural abscess above the tegmen tympani may infect the superior petrosal sinus or a petro-squamous sinus, and the infection may be conveyed into the sigmoid flexure of the transverse sinus (Körner). It very often extends to the bulb of the jugular vein. We speak of a primary thrombosis of the bulb when the infection passes directly from the tympanic cavity to the bulb of the jugular vein. This occurs chiefly in children (M'Kernon). Traumatic sinus thrombosis mainly affects the region of the superficial sinuses, and the form which is due to inflammatory processes in the face, orbit, etc., is usually localised in the cavernous sinus.

Here the infection commonly passes through the vena anguli nasi and the superior ophthalmic vein to the cavernous sinus. See Nonne, *N. C.*, 1904, and the following discussion.

Symptomatology.—We seldom have an opportunity of studying the symptoms of pure sinus thrombosis. In marantic cases they are partially masked by the primary disease and the symptoms of brain anæmia, and in secondary thrombosis by caries and complications (meningitis, abscess). The symptoms which are really most characteristic, viz., those directly due to occlusion of the sinus, are marked only in a few cases. These consist of *signs of obstruction* in the region of the external *veins of the skull* and the *veins of the face* which are connected with the sinus or open into it. Thus thrombosis of the cavernous sinus sometimes causes dilatation of the *frontal veins*, cyanosis of the orbital and frontal regions, swelling of and around the eyelids, and *protusion of the eyeballs* [1] from dilatation of the retrobulbar veins. In rare cases there have been symptoms of *obstruction in the retinal veins*, and *choked disc* has even been noted, but Jansen has never observed it in his cases. Uhthoff also states that typical choked disc is uncommon in sinus thrombosis, especially in the marantic form. A short time ago Jansen and I observed a case of sinus thrombosis in chlorosis, in which the choked disc was exceedingly severe (with hæmorrhage, etc.). Hawthorne also found this in another case. Amblyopia and amaurosis have been

[1] The frequency of exophthalmos in thrombosis of the cavernous sinus has been specially noted by Uhthoff.

often observed, and I have once seen them along with a normal fundus of the eye. Thrombosis of the central retinal vein has also been described. In many cases there is *neuralgiform pain* in the area of the first branch of the trigeminus, and symptoms of paralysis appear in the muscles supplied by the *abducens*, *oculo-motor* and *trochlear* nerves (which pass through the wall of the sinus), but Jansen has found these to be less common than one would have expected.

Thrombosis of the *transverse sinus* is indicated in many cases by *œdematous swelling* of the soft parts behind the mastoid process (Griesinger) and by dilatation of the cutaneous veins. Palpation often reveals an extension of the thrombus into the first portion of the jugular vein; Jansen, for instance, found that this vein was involved in about a third of all the cases of otitic sinus thrombosis. The vein is tender, both spontaneously and on pressure; it feels like a firm, tender column; the soft parts and glands in its neighbourhood are swollen, there is pain in the side of the neck on movement, especially on rotation of the head, torticollis, pain on swallowing; swelling and tenderness of the retro-maxillary fossa (R. Hoffmann). Phlebitis of the jugular vein may, however, be simulated by lymphangitis and burrowing abscess. Gerhardt has drawn attention to the fact that the external jugular vein is less full on the affected than on the healthy side (because it can more easily give off its blood into the less congested internal jugular vein). Jansen did not find this inequality in the fulness of the jugular veins of the two sides, and, indeed, this sign is usually absent.[1] Zaufal describes dilatation of the veins of the mastoid foramen. The percussion-dulness over the mastoid process (Okukeff) is due to swelling of the soft parts (Thies-Barth) and is an uncertain sign. Symptoms of paralysis in the glosso-pharyngeal, vagus, accessory and hypoglossal nerves have been observed, but they are exceedingly rare.

Kümmel has noted pain on swallowing in thrombosis of the bulb of the jugular vein, and he attributes it to neuritis of the glosso-pharyngeal nerve.

There is in many cases of thrombosis of the *longitudinal sinus* excessive fulness and sinuosity of the veins in the frontal, parietal and temporal regions (the caput medusæ of Lermoyez), and œdematous swelling in these zones. *Nasal hœmorrhage* has often been observed; it is due to excessive fulness in the nasal veins. In children the fontanelles are distended and protruding on account of the congestion in the cerebral veins and the increase of intracranial pressure.

These diagnositc symptoms are very inconstant, and the usual signs of sinus thrombosis are of an *indefinite* nature. They are in many respects identical with those of meningitis and focal diseases, with the latter as a matter of course, since *softenings* and *hœmorrhages* in the brain substance are almost constant results, and *abscesses* are common complications of this disease.

In carefully observed cases of autochthonous sinus thrombosis (Gutheil, Bücklers, Reinhold, Ehrendorfer, Voss, Good, Spiller-Camp, etc.) the following symptoms have been noted: The patient, either suddenly, or after having suffered from chlorosis, hydrocephalus, phthisis, etc., became affected with violent *headache*, followed by *vomiting*, *stupor*,

[1] A still more uncertain sign is that described by Voss, namely, that the murmur caused by pressure of the stethoscope is absent on the affected side.

which soon developed into *coma*, *delirium*, general or unilateral *convulsions* and *paralysis*. Disturbances of co-ordination, contracture, tremor, choreic tremors, etc., were less common. In a few cases there was *rigidity of the neck*, but seldom rigidity in the muscles of the extremities. The paralysis affected the extremities of one side of the body, or in exceptional cases, *e.g.* that described by Pineles, all four extremities. The cranial nerves were seldom paralysed, the oculo-motor being most apt to be involved. Aphasia and hemianæsthesia have occasionally been noted. Choked disc was present in a few cases (Hoffmann, Voss, Oppenheim), and amaurosis without opthalmoscopic changes, or simple venous congestion of the retinal veins, have been observed by Good. The *temperature* was usually normal, but sometimes raised; an abrupt rise of temperature has specially been noted, and in a few cases it rose just before death above 42° C. The pulse rate varied; it was usually normal or slow to begin with, but became more rapid towards the end. The respiration corresponded somewhat to the pulse; in the last stage it sometimes assumed the Cheyne-Stokes character. As a rule the patient died in a state of *coma* at the end of a few days; or much more rarely after one to two weeks or even months (Voss).

From this description it is obvious that the diagnosis can in such cases be made only from the cause and the symptoms of congestion which may be present (possibly also from the presence of venous thrombosis at the peripheral parts of the body); the general symptoms would point rather to meningitis or to some focal disease. The latter are actually present in the form of foci of hæmorrhage and softening in the hemispheres. They were absent only in one of the cases described by Nonne,[1] although the presence of cortical epilepsy, hemianæsthesia and hemianopsia made it impossible to doubt the existence of a widespread focal disease. If the external signs—symptoms of congestion, etc.—are absent, a provisional diagnosis only can be made, and in such cases a confusion with meningitis, cerebral hæmorrhage, encephalitis, etc., is always possible and excusable. A case published by Luce[2] seems to show that thrombosis of the longitudinal sinus may follow empyema of the antrum of Highmore, but the relationship is not very clearly explained. The difficulty of diagnosis is shown clearly by Wimmer's[3] case. Another circumstance enters into the differential diagnosis, viz., that signs of a severe brain disease of which as yet we do not definitely know the organic cause may develop in the course of chlorosis (Immermann, Eichhorst, Müller, Burton-Fanny, etc.). A similar group of symptoms may be produced in the hydrocephaloid of childhood by exhaustion and cerebral anæmia.

The diagnosis of secondary phlebitic, and especially of otogenic, sinus thrombosis is not a matter of so much difficulty.

Uncomplicated thrombosis of the transverse sinus in chronic suppuration of the middle ear, if it is not septic and if the flow of the blood is not cut off by a solid thrombosis, does not necessarily give rise to any symptoms other than slight fever and headache (Jansen), and even these may be absent (Herzfeld).[4] As a rule, however, it gives rise to symptoms,

[1] *Mitt. aus d. Hamb. Staatskr.*, 1897.
[2] *N. C.*, 1904; see, however, Fraenkel's criticism in the following discussion.
[3] *B. k. W.*, 1906.
[4] *Z. f. Chir.*, Bd. xlix. See also Schröder, *Z. f. Ohr.*, Bd. lii.

some of which resemble those in meningitis, brain abscess, and extradural suppuration, but some of which are characteristic and even pathognomonic. Extradural abscesses round the sinuses are very often the immediate cause, and as their symptoms consist in the usual prodromata of thrombosis, they should receive careful consideration. In any case they indicate the danger which is threatening. Jansen mentions the following symptoms of abscesses of this kind: protrusion of the bones, subperiostal abscess and phlegmon behind the mastoid process, especially round the mastoid foramen and on the adjacent part of the occiput and also on the posterior segment of the mastoid process itself, pain on pressure and percussion on these parts, limitation of the movement of the head, especially on the sagittal axis, torticollis, nystagmus, etc. There are also a number of symptoms which may be due either to extradural suppuration or to thrombosis. These include optic neuritis and choked disc,[1] which, however, is much more frequent in sinus thrombosis (34-45 per cent. of all the cases) than in extradural abscess, and also headache (although this may be absent (Herzfeld)), vertigo, vomiting, slow pulse, stupor, excitability, tenderness of the spinous processes to pressure, rigidity of the neck, and other meningeal symptoms of irritation.

If signs of *pyæmia* or jugular phlebitis and periphlebitis appear, the diagnosis is certain. Thus pyæmic fever—remittent fever rising abruptly to 40-42° and falling to considerably below normal — acceleration and irregularity of pulse, repeated rigors, abundant perspirations, diarrhœa, jaundice, enlargement of spleen, metastases (especially pulmonary — pulmonary infarct, pulmonary abscess), and less often metastatic abscesses of the joints, etc., are specially characteristic; they indicate that the process has extended to the sinus and that the septic matter has made its way into it. Jansen, Hessler, O. Brieger, Körner, Leutert, and Goldschmidt have thoroughly discussed these forms of pyæmia of otogenic origin.

Takabatake has published special articles on pyæmic fever (*Z. f. Ohr.*, Bd. xlv.).

Distinct evidence is also afforded by the local *external* signs of sinus thrombosis (see above), especially by those which are caused by involvement—phlebitis and thrombosis—of the jugular vein. Abscesses may also develop in the region of the mastoid and condyloid veins, and less frequently in the deep veins of the neck. Symptoms may occasionally appear which are caused by compression of the glosso-pharyngeal, vagus, and accessory veins (less often of the hypoglossal), and this may give rise to a slowing of the pulse, which does not otherwise occur in secondary sinus thrombosis.

Spinal symptoms, such as flexion contracture of the lower extremities, paraparesis, and Westphal's sign, have occasionally been observed in sinus thrombosis. In such cases the thrombosis was associated with serous meningitis. It should always be remembered, however, that otitic sinus-thrombosis, especially in children, may throughout its whole course resemble meningitis, even when it is not complicated by this disease. In sinus-thrombosis, however, there is, as a rule, no increase in the cerebro-spinal fluid (Körner). Pyæmic symptoms may also

[1] Out of sixteen cases of unilateral phlebo-thrombosis of the transverse sinus without intracranial complications Körner found only two with opthalmoscopic changes. See also Uhthoff, *loc. cit.*, and *M. f. P.*, xxii.

represent the clinical picture, all the local signs being absent, for there is no doubt that an otogenic pyæmia may occur *without* sinus-thrombosis. Körner attributes it to phlebitis of the small veins of the mastoid process which open into the transverse sinus (pyæmic osteo-phlebitis). The course of the septic form is practically the same as that of the pyæmic. Finally, we must remember that thrombosis is very often associated with suppurative meningitis and brain abscess. Lumbar puncture may show whether the thrombosis is accompanied by a suppurative meningitis or not (Leutert).

Thrombosis may extend from the transverse to the inferior petrosal and *cavernous* sinuses. The latter may also be directly affected, and as a rule in this case the affection passes from the cavernous sinus of one side to that of the other (Macewen). The process may extend from the ear to the bulb of the jugular vein alone, or one of the small sinuses of the petrous bone may be also affected, but this is unusual.

The chapter on meningitis and brain abscess should be consulted with regard to differential diagnosis. Brain symptoms which simulate thrombosis may also be caused by toxæmia due to suppuration on the skull or in the parietal bone (Eulenstein, *Z. f. Ohr.*, Bd. xl.). Mann also attributes some of the symptoms which appear in intracranial complications of otitis to toxic influences ("Ber. d. Ges. f. Nat. u. Heilk.," Dresden, 1901-02). In one case described by Chauffard (abs. *R. n.*, 1904) the sinus thrombosis was masked by a psychosis. Hastings reports confusion with malaria (*Journ. Amer. Med. Assoc.*, 1905). With regard to differential diagnosis, consult also Voss (*Z. f. Ohr.*, Bd. l.).

The *prognosis* of sinus thrombosis is unfavourable. We are not, however, justified in calling the disease incurable. In rare cases of primary thrombosis there has been spontaneous recovery, probably due to absorption of the thrombus, but even when the thrombosis is persistent it does not necessarily lead to permanent impairment of the circulation. There has occasionally been recovery with persistence of the focal symptoms. Recovery is rarer in phlebitic thrombosis, but cases have been reported in which there has been *recovery from pyæmia following thrombosis of the sinuses* (Schwartze, Schulze,[1] etc.).

In infective thrombosis the danger arises mainly from pyæmia and suppurative meningitis. The danger of pyæmia is greater in thrombosis of the transverse and inferior petrosal sinuses than in thrombosis of the superior petrosal and cavernous sinuses, on account of the direct communication of the former with the jugular. If the thrombosis extends from the transverse sinus far towards the torcular Herophili, meningitis almost always develops (Jansen). Körner thinks that in the pyæmic form the prognosis is fatal if the pulse does not slow down as the temperature falls.

The prognosis of secondary thrombosis has become much more favourable of late years owing to *surgical* treatment. There is no prospect of recovery when the case is complicated with diffuse suppurative leptomeningitis, and but little in circumscribed suppurative leptomeningitis, brain abscess, and extensive pyæmic metastases in the lungs, etc., but operation has led to recovery in a few cases of commencing pyæmia.

Metastatic pulmonary abscesses have also been cured by operation (Alt [2]).

Treatment.—In cases of marantic thrombosis of the sinuses, the

[1] *A. f. Ohr.*, Bd. liii.

[2] *W. m. P.*, 1902.

treatment is symptomatic. We should try to improve the condition of the circulation as far as possible, to strengthen the heart by the administration of stimulants, nourishing food, etc. Blood-letting is generally contra-indicated. For the headache an ice-bag may be applied, or narcotics given, but blood-letting is not suitable. The patient should lie upon his back, with his head slightly raised.

In cases of phlebitic thrombosis care should be taken that the pus can flow freely away. The more completely the pus is drawn away from the neighbourhood of the sinus, the greater is the prospect of preventing septic disintegration of the thrombus. Much may be done in the way of prophylaxis by careful treatment of the primary disease (incision of the carbuncle, of the infiltrated parotid, *trephining of the mastoid process*, *evacuation of extradural abscesses*, etc.).

Hildebrandt (*B. k. W.*, 1904) has seen the symptoms disappear in a case of rhinogenic thrombosis of the cavernous sinus after the use of antistreptococcic serum.

Surgical treatment has of late years made great advances in this direction. Zaufal first pointed the way (1880), and subsequently Lane, Ballance, Horsley, Parker, Salzer, Bacon, and Macewen, and Jansen in particular, have shown that life can often be preserved by early operation, cutting down upon the sinus and clearing out its contents. Macewen was successful in 20 out of 27 operations, and Jansen in 11 out of 24. Hensche, who has collected the statistics of Macewen, Chipault, Jansen, and Körner, found that recovery had taken place in 85 out of 145 cases, therefore in 58 per cent. Brieger, Block, Herzfeld, Grunert, Joung, Alt, Hildebrandt, Mann, Sturm-Sucksdorf,[1] Voss,[2] Kümmel, and others have also reported cures. Körner's latest statistics show 28 recoveries in 38 cases—74 per cent. Jansen thinks that the existence of an affection of the wall of the sinus, or even of a solid thrombus, does not in itself indicate an operation, as these affections often disappear spontaneously. On the other hand, the sinus should be opened (1) when there is evidence of a septic disintegrating thrombus, (2) in cases of gangrene of the wall of the sinus, (3) when there are repeated rigors, a grave general condition, optic neuritis, etc. Opinions differ as to whether the internal jugular vein should be ligatured or not, in order to prevent the development of pyæmia. Zaufal, Körner, and others advocate this measure, whilst Jansen thinks it is not called for when the thrombosis is limited to the sinus.

In his last discussion of this question Jansen states that ligature of the jugular vein is indicated *before* an operation upon the sinus: (1) in undoubted phlebitis of the jugular vein; (2) in severe sepsis; and *after* exposure of the sinus: (1) when the latter appears to be healthy, when there is no suppuration outside the sinus, and when the pyæmia is accompanied by marked oscillations of temperature and rigors; (2) in periphlebitis or thrombosis of the walls accompanied by the same conditions. The jugular vein should be ligatured after the sinus is opened: (1) when the septic thrombus lies in the immediate neighbourhood of the bulb; (2) when opening of the sinus is not followed by cessation of the rigors and fall of the temperature, etc. We need not here discuss the views of Brieger, Leutert, etc., which somewhat differ from those of Jansen. See also Grünert ("Die operative Ausräumung d. Bulb. venæ jug. bei ot. Pyämie," Leipzig, 1904; *A. f. Ohr.*, Bd. lvii.); Stenger (*A. f. Ohr.*, Bd. liv.); Voss (*B. k. W.*, 1904, and *D. m. W.*, 1904); Randall (*Journ. Amer. Med. Assoc.*, 1904); and the discussion of Hastings' paper (*Journ. Amer. Med. Assoc.*, 1905). Körner describes the technique of the operation (*loc. cit.*).

Definite evidence of the existence of diffuse, suppurative meningitis is a contra-indication to

[1] *Z. f. Ohr.*, Bd. xli.

[2] See bibliography in Körner.

operative treatment of sinus thombosis, but the presence of pus or cocci in the cerebro-spinal fluid should not of itself preclude operation (Jansen).

Thrombosis of the longitudinal sinus affords an opportunity for operation in a few cases only. The danger that air may get into the sinus, and so give rise to an air embolism, is slight. This symptom has seldom been observed, even in other injuries of the sinus. Kuhn reports one such case, and Körner has collected a few others.

Cerebral Tumour

The most important literature on this subject is for the most part to be found in the following monograph and treatises: Bernhardt, "Beiträge zur Sympt. und Diagnost. der Hirngeschwülste," Berlin, 1881; Bramwell, "Intracranial Tumours," London, 1888; Bergmann, "Die chir. Behandl. der Hirnkrankheiten," 3rd edition, Berlin, 1899; Oppenheim, "Die Geschwülste des Gehirns," 1st edition, Vienna, 1896; 2nd edition, Vienna, 1902; Bruns, "Die Geschwülste des Nervensystems," 1st edition, Berlin, 1897; 2nd edition, 1908; Chipault, "Chirurgie opér. du Syst. nerv.," 1894; Auvray, "Les tumeurs cérébrales," Paris, 1896; Duret, "Les tumeurs de l'Encéphale," Paris, 1905; Mills, Frazier, and Spiller, "Tumours of the Cerebrum," etc., Philadelphia, 1906; Mills, Frazier, Schweinitz, etc., "Tumours of the Cerebellum," New York, 1905; Mills, *Univ. of Penn.*, 1906; Oppenheim, "Beitr. zur Diagnost. und Therap. der Geschwülste," etc., Berlin, 1907; Ballance, "Some Points in the Surgery of the Brain," etc., London, 1907; 2nd edition, 1908; Horsley, "On the Technique of Operations in the Central Nervous System," *Brit. Med. Journ.*, 1906.

The brain is the favourite site of new formations. There is hardly any kind of tumour which does not occur in it. The most frequent forms of brain tumour are *glioma*, *solitary tubercle*, *sarcoma*, and *gumma*. *Carcinoma* is somewhat less common, and pure *fibroma*, *angioma*, *lipoma*, *choleasteatoma*, *psammoma*, and *osteoma* are very rare. Neurofibromata, so called (see p. 596), may also involve the cranial nerves, especially the auditory and vagus. Papillomma, cylindroma, and dermoid cysts must be regarded as rarities.

Mixed forms of glioma and sarcoma are not unusual; as least they are often described. The glioma may assume a myxomatous character. Osteosarcoma, fibrosarcoma, and angiosarcoma are also occasionally found in the brain.

In the adult, glioma, syphiloma, and sarcoma are the most common brain tumours, and in childhood tubercle is the most frequent. But all these forms may occur at any age, although tubercle is rarely found after the age of thirty.

Glioma may be any size from that of a hazel-nut, a hen's egg, to that of a fist. It is not sharply defined from the surrounding brain substance, and it does not extend by pressure, but by *infiltration* of the brain tissue, so that it cannot as a rule be simply shelled out. A section through the tumour shows a yellow-white or grey-red colour; it sometimes differs very little in its tint from the normal grey or white matter, but is usually mixed with grey-red, gelatinous, and hæmorrhagic portions, which gives the section a variegated look. If large hæmorrhages have taken place within the tumour, these may so mask the tumour-character that an inexperienced observer would diagnose a hæmorrhagic softening. On close examination, however, islands of new-formed tissue will always be found, especially in the peripheral parts. Cystic degeneration may also cause the tumour to be unrecognised, but in such cases there is always a mantle of tumour tissue in the periphery. Cavities with a cell lining are rarely found in glioma. According to Ströbe, these arise from an abnormal budding at an early period of embryonal life of the neural tube, *i.e.* which later becomes the ventricle, and he thinks the glioma takes its rise from these pre-formed cavities. The relations are therefore similar to those which we assume between syringomyelia

and gliosis (see p. 377). Jores has also explained the conditions which he found in this way, whilst Henneberg and Saxer (*Ziegl. Beitr.*, xxxii.) are not in favour of Ströbe's view.

The glioma consists mainly of *glial cells* and an intercellular fibrillar net-work. We do not exactly know whether the fibres are or are not exclusively cell-processes. True Deiters' cells are also numerous in glioma, and many varieties of glial cells (giant cells and structures like nerve cells) have been found (Pels-Leusden, Stroebe). The tumour process may also give one the impression of a local hypertrophy; there are indeed tumours of this and similar kinds which cannot be clearly recognised by the naked eye, the tumour tissue being revealed only by microscopic examination (Oppenheim, Nonne-Stertz). Marburg (*Obersteiner*, xiii.) has lately related some remarkable facts as to the relation of glioma and so-called brain hypertrophy. Glioma originates in the brain itself, and affects neither the cerebral membranes nor the bones.

Sarcoma, on the other hand, arises preferably from the meninges, the periosteum, and the bones, and may grow in the direction of the cavity of the skull. Even when it is of a great size, it may simply compress and displace the neighbouring parts of the brain. But it also often penetrates into the brain substance, or it may develop within it. Even in such a case it is usually sharply defined, and is separated from the healthy brain substance by a zone of softening. These new-growths vary greatly in size, from that of a nut or pigeon's egg to that of a fist, or may be larger. The fibrosarcomata arising from the periosteum are sometimes unusually large. On the whole the consistence of a sarcoma is firmer that than of a glioma. Caseation and breaking down of some parts of the tumour from conversion into fat has occasionally been observed. It may be impossible by naked-eye examination to distinguish glioma from sarcoma. A diffuse sarcomatosis may be present in addition to tumours in the meninges of the brain and cord. Indeed, sarcoma may entirely lose its tumour character, and may extend through the brain and cord in the form of a *diffuse meningeal process which can only be recognised by microscopical examination*, as Nonne, Rindfleisch, Redlich, Barnes, and Grund (*Z. f. N.*, xxxi.) have shown. Both round cell and spindle cell sarcoma have been found in the brain. A sarcomatous neuritis of the cranial nerves has also been described (Darkschewitsch, Klimow).

Glioma may also arise from the ependyma of the ventricle (see Hildebrandt, Inaug Diss., Berlin, 1906). Tumours of the ventricles have sometimes the character of sarcoma or carcinoma; the latter usually arises from the choroid plexus.

Endothelioma is the name given to tumours which arise from the endothelia of the spaces of the connective tissue and from the lymph and blood vessels of the meninges. The endothelioma of the dura mater consists, according to Birch-Hirschfeld, of connective tissue rich in vessels, the meshes of which contain flat cells, concentrically arranged. In the pia they develop partly in the form of nodules, and partly of flat patches. These tumours can hardly be definitely distinguished from sarcoma. They have also been described as alveolar sarcoma, endothelial sarcoma, and perithelioma.

Carcinoma is a soft and very vascular tumour of irregular form. It sometimes spreads in a superficial manner over the dura, and sometimes assumes the form of well-defined or diffuse tumours within the brain. Wernicke says that it may be as large as a child's head, and he emphasises the tendency to pulpy softening and to destruction of tissue. Carcinoma rarely appears in the brain as a primary tumour, but the metastatic form is common. A case described by Buchholz (*M. f. P.*, iv.) shows the extraordinary multiplicity and extent which metastatic carcinoma may assume in the brain. It should be remembered that a cell infiltration of the pia, which is only revealed by microscopic examination, may also occur in carcinoma (Saenger). Metastatic carcinoma may extend in this diffuse manner throughout the meninges of the brain and spinal cord (Siefert, *A. f. P.*, xxxvi.).

Syphiloma and *solitary tubercle* have much external resemblance. They are usually small tumours of the size of a hazel-nut or a walnut, but a tubercle may become almost as large as a goose's egg (see Fig. 329, Pl. vi.). Neither of these forms are very well supplied with vessels, and they have a tendency to caseation. Tubercle is apt to undergo suppurative softening, and may exactly resemble an abscess, as in the case illustrated by Fig. 329. The younger, peripheral portions of the new-growth show the characteristics of granulation tissue. An eruption of miliary tubercle into this zone, or into its neighbourhood, is characteristic of solitary tubercle. A further guide to the differentiation is afforded by the fact that gummatous tumour almost always arises from the *meninges*, and its connection with these can be distinctly recognised. Its tendency to spread flatly over the surface is particularly remarkable. There is also, however, a localised tubercular meningoencephalitis which has a similar distribution. Bacteriological

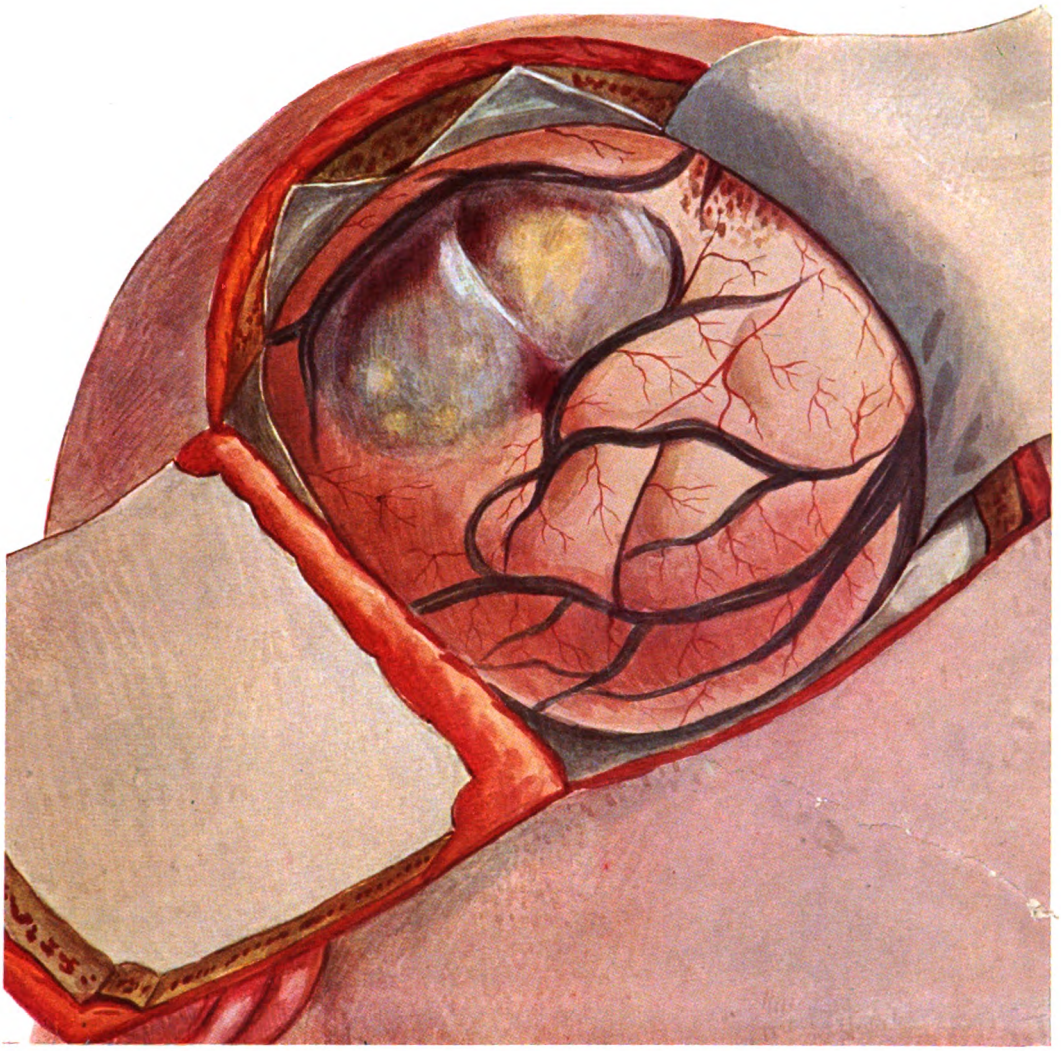

Fig. 329.

Tubercular Tumour in Parietal Region.

(From a sketch made during operation.)

Otto Schulze & Co., Edinburgh.

examination affords the most certain criterion for the differentiation of these two kinds of tumour; *tubercle bacilli* are common in solitary tubercle, and the spirochæta pallida in syphiloma. *Actinomycosis* is a form of new-growth very seldom found in the brain, and metastases from *deciduoma* malignum are also very rare (Marchand, Siefert, *A. f. P.*, Bd. xxxviii., etc.).

Psammoma, which occurs almost exclusively in the meninges and the pineal gland, but which may also be found in the cerebrum, is a new-growth containing abundant chalk-concretions, which often show no tendency to growth. *Adenoma* originates in the hypophysis.

Cholesteatoma is often found incidentally during post-mortem examination, but it may in some cases give rise to all the symptoms of cerebral tumour. The term " pearly tumour " is given to it from the white, shining pearls, which give it a mother-of-pearl appearance. These new-growths probably originate in scattered nuclei of epithelium (Boström, Bonorden, Ribbert, Ziegler, Benda, E. Meyer). They are found chiefly at the base and the median areas of the brain (region of the tuber cinereum, of the corpora mamillaria, the corpus callosum, the choroid plexus of the ventricle, the space between the pons and oblongata, etc., see Fig. 330). Other writers (Birch-Hirschfeld) include cholesteatoma among the endothelial neoplasmata. A detailed

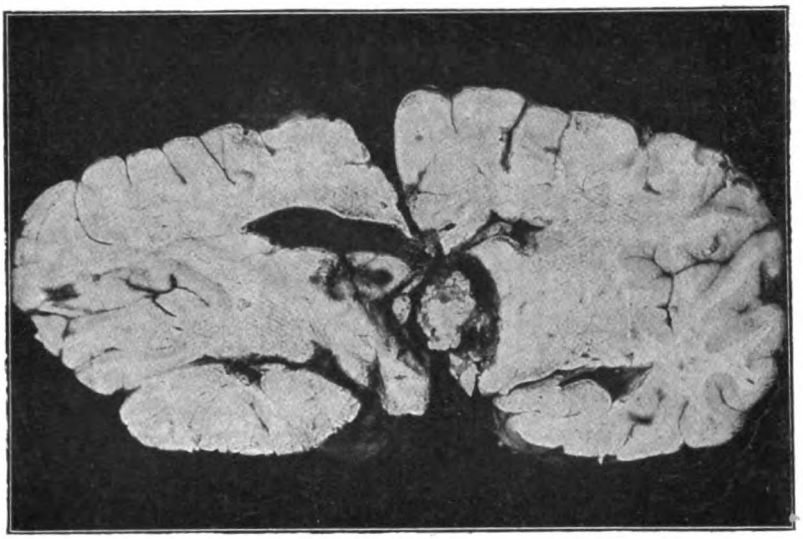

FIG. 330.—Cholesteatoma of the base of the brain in the region of the third ventricle. (Oppenheim.)

description of this kind of tumour will be found in Erdheim (" Über Hypophysenganggeschwülste und Hirncholesteatome," *Sitz. d. k. Akad. d. W.*, Wien, 1904) and in Scholz (*V. A.*, Bd. clxxxiv.).

Angioma appears in the brain in various forms. It may appear as a circumscribed, encapsulated structure, resembling an angioma cavernosum (Bruns, Oliver, Struppler, Bielschowsky, etc.), or a diffuse, usually meningeal new-growth of the character of teleangiectasia or of angioma arteriale racemosum. This form has been described by Kalischer (*A. f. P.*, Bd. xxxiv.), Emanuel, and Bergmann-Oppenheim (see Fig. 331). As regards angioma racemosum and serpentinum, consult Simmonds (*N. C.*, 1905).

Cysts of the brain are either of parasitic origin (cysticercus, echinococci, and the varieties of distomum which occur in Japan) or they are degenerated neoplasms. The origin of traumatic cysts is not yet clearly explained. They occur chiefly in the mantle of the cerebrum and in the cerebellum. Their symptoms may be very similar to those of tumour. We leave out of account here the cysts arising from hæmorrhage and softening and from encephalitic foci, but clinically these cannot be definitely distinguished from true cysts, and they have repeatedly given occasion for operative treatment. It is not yet proved whether a localised serous meningitis may give rise to the formation of cysts, as Thiem and Cramer have assumed in one case. A case described by Placzek-Krause (*B. k. W.*, 1907) also points to this probability. Finally, there are cysts

produced by anomalies of development, and the formation of diverticula of the cavities of the brain (Virchow). Cases of this kind have been reported in recent literature (Herzog, Schüle, Bland Sutton, Bielschowsky). Lichtheim (*D. m. W.*, 1905), Scholz (*Mitt. a. d. Grenzgeb.*, xvi.), Auerbach-Grossmann (*Mitt. aus d .Grenzgeb.*, xviii.), and F. Henschen (*Z. f. k. M.*, Bd. xciii.) have also made important contributions to this subject. In a few cases, such as one treated by Krause and myself, the cysts have no walls ; in our case these cysts were situated symmetrically in both cerebellar hemispheres. The cystic content is sometimes watery and serous, sometimes viscous and colloid-like, and of a yellow or lemon colour. As a rule the fluid differs from cerebro-spinal fluid in the greater amount of albumen which it contains.

Ziehen (*N. C.*, 1906) and Raymond (*R. n.*, 1906) have lately reported cases of the rare dermoid-cysts in the brain.

Peculiar mixed forms of different kinds of tumours, such as glioma and tubercle, or the development of a tubercle within the gliomatous new-growth, or some other combination, have been observed in some rare cases (Reich, Kazowsky, Warthin). Cone states that he has seen a combination of carcinoma, sarcoma, endothelioma, glioma, and tubercle within a neoplasm (?).

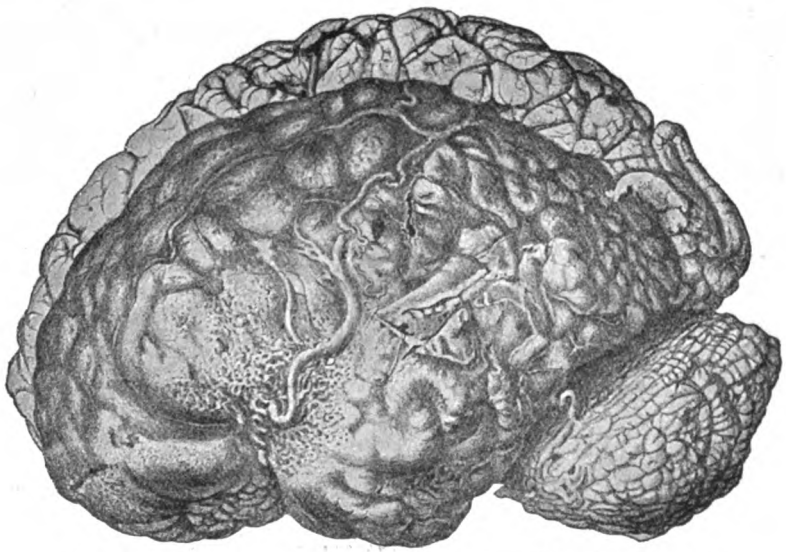

FIG. 331.—Diffuse angioma of the meninges. (After S. Kalischer.)

Behrendsen describes the simultaneous appearance of growths of two different kinds within the cranial cavity (cholesteatoma of the pia and glioma of the hemisphere).

In rare cases benign growths of other organs may lead to malignant metastases in the skull or brain. Such cases have been described by Flatau-Kölichen and by Borrmann (*Ziegl. Beitr.*, Bd. xl.).

Site of the tumour.—Tumours may arise from any part of the brain, but its various regions are not equally liable to become affected. The majority of tumours develop in the cerebrum, especially in its centrum ovale. The region next most often affected is the cerebellum ; then follow the pons and the central ganglia, then the corpora quadrigemina, etc. Certain relations may be established between the character of the tumour and its topography. Thus glioma affects chiefly the hemispheres of the cerebrum, the cerebellum, and the pons, and only rarely appears in other parts. Solitary tubercle prefers the pons, the cerebellum, and the cerebral cortex. Syphiloma is very seldom found in the cerebellum,

and still more rarely in the central ganglia. Sarcoma most commonly makes its way into the cavum cranii from the periphery and from the meninges; the bones of the base of the skull, the parietal bone, the sphenoid bone, and less often the occiput are its sites of origin.

Other characteristics (rapidity of growth, multiplicity, etc.).—Glioma grows very slowly, and not infrequently becomes arrested. It grows rapidly larger only when there is great hæmorrhage in the tissue of the tumour. Soft sarcoma increases in size more quickly than firm. Osteo-fibro-sarcoma, osteoma, angioma, and cholesteatoma are particularly slow of growth. Carcinoma increases very rapidly. Tubercle may grow quickly, but it may also remain stationary for a long time and may undergo regressive changes. Suddenness of proliferation and of degenerative changes are specially characteristic of syphiloma.

Glioma is, as a rule, a single tumour, and so usually is sarcoma, but a multiple development of sarcomata, or a combination of one or more tumour nodules with diffuse sarcomatosis, has been observed in several cases (Ollivier, Schultze, Schateloff, Hippel, A. Westphal, Nonne, Lereboullet, etc.). Glioma is always of a primary nature, whilst sarcoma is more often of metastatic origin. Carcinoma of the brain, which is usually of a metastatic nature (primary tumour in the mamma, lungs, pleura, etc.), is seldom a solitary tumour. Multiplicity is also the rule as regards syphiloma and tubercle. In 100 out of 185 cases there were multiple tubercles in the brain. It should not be forgotten that these are post-mortem conditions, and that the solitary occurrence of a tubercle is certainly not unusual at the beginning or in the earlier stages of the disease.

We have now to consider what effect the development of a new-growth in the brain has upon the whole organ and its neighbourhood? In making an autopsy we can usually see as soon as the skull has been opened that a neoplasm has made room for itself in the brain. The gyri are almost always flattened, the sulci effaced; it is evident that some pressure from within has pushed the cortex outwards. The pia and the brain itself look dry, and usually contain very little blood. Trephining during life shows as clearly, or even more so, that the dura is very tense and bulging, and that it usually does not pulsate. After the dura has been cut open, the brain protrudes through the opening. The changes caused by the increased pressure affect specially that part of the brain in which the tumour is situated. These changes are usually absent if the tumour arises from the cortex itself or from the membranes of the brain. The tissue in the neighbourhood of the tumour is usually softened.

Marburg and Bielschowsky (*Journ. f. P.*, vii.) have made remarkable investigations as regards the histology of the nervous substance affected by the tumour, especially of the axis-cylinders. See also Weber-Papadaki (*Nouv. Icon.*, xviii.).

The parts of the brain which are subjected to pressure from the tumour undergo various displacements and changes in form. Thus in one of my cases of tumour of the cerebello-pontine angle (Fig. 332), the corresponding half of the pons had been compressed into a quarter of its size. I have lately seen this in a number of cases (*e.g.* Fig. 333). The falx cerebri may be greatly displaced to the side, parts of the cerebellum may be pressed into the foramen magnum, etc.

The cerebro-spinal fluid is almost always increased. *Internal hydrocephalus* tends to be very marked in tumours of the cerebellum and the corpora quadrigemina, as the tumour by its pressure cuts off the communication between the ventricles, or gives rise to venous obstruction and increased transudation by compression of the veins (vena magna Galeni) which convey the blood from the choroid plexus into the sinus rectus. The hydrocephalus is seldom limited to the cerebral ventricle of one side or to one ventricle.

The irritation exercised by the tumour upon the brain may give rise to general swelling and increase in its size, and in cases of long duration this may be followed by diffuse structural changes (glial proliferation). The disproportion between the capacity of the skull and the weight of the brain may also give rise to symptoms, as Reichardt has shown (*Z. f. N.*, Bde. xxvii. and xxviii.).

The most important points with reference to the causation of "choked disc" have already been mentioned on p. 715. The *nerves* at the base

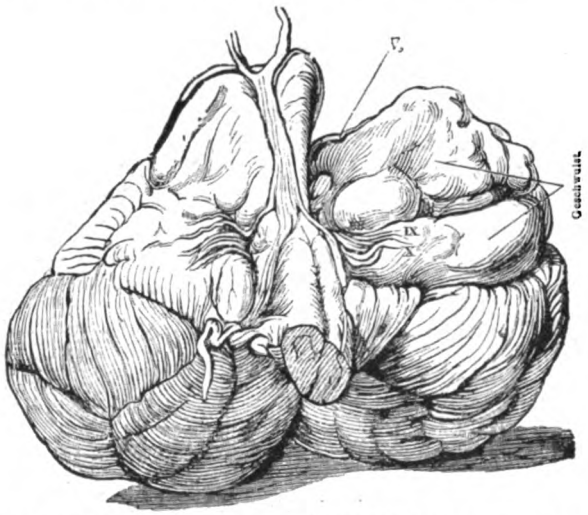

FIG. 332.—Cerebellar tumour, with atrophy of the pons due to pressure. (Oppenheim.)

of the brain are not infrequently flattened and compressed, either directly by the tumour or as the result of the general increase of intracranial pressure. This is specially marked when severe hydrocephalus is present in addition to the tumour. This flattening is found in the oculomotor, but still more often in the abducens and olfactory nerves. The nerves may also undergo strangulation from the basal arteries, which are dragged by the tumour and raised from the underlying parts (Türck, Sachs, Erdheim, Bartels[1]). The floor of the third ventricle, which is distended by hydrocephalus, may directly compress the chiasma. Finally, the bones of the skull itself undergo certain changes. When the tumour is a superficial one, *osteoporosis* not infrequently develops on the neighbouring parts of the skull. But even when the tumour lies deep down in the medulla, the general increase of pressure may give rise to an osteoporosis which extends over the whole skull (especially the roof). Perforation, however, is exceedingly rare, if we except those tumours which are

[1] *Z. f. Aug.*, xvi.

situated upon the periosteum and the dura and which grow in an outward direction. Tumours of the hypophysis press towards the sella turcica, which may become very much bulged and necrosed, as X-ray examinations have lately proved (see below). Increase of the brain pressure in childhood may cause the sutures which have already closed up to burst open. In a case of this kind, in a child suffering from rickets, I have found marked hyperostosis of a tumour-like character at the site of the sutures, and especially in the region of the large fontanelles. The base of the skull is the usual site of hyperostoses of the skull in rickets (Chiari, Regnault, Homén). In a few cases rupture of the sutures of the skull occurred at the age of 13 to 15, and even later (Jackson, Steffen, Anton, Wollenberg).

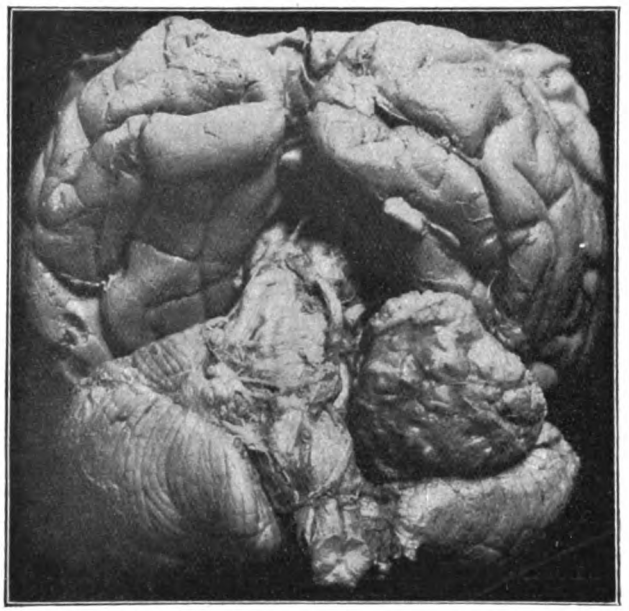

FIG. 333.—Neurofibroma of the right acoustic nerve. Death after commencement of operation. (Oppenheim-Krause.)

Changes in the spinal cord, and chiefly in the posterior roots and columns, have lately been found in cases of brain tumour, especially in tumour of the cerebellum (Dinkler, Mayer, Anton, Pick, Hoche, Ursin, Finkelnburg, Kirchgässer, Becker, Erbslöh, Nageotte, Raymond). These are probably due to the increase of pressure exerted by the tumour upon the cerebro-spinal fluid. This view is specially advocated by Batten and Collier (*Br.*, 1899) as the result of their very careful investigations. They state that brain tumour gives rise, in about 65 per cent. of the cases, to degeneration of the posterior columns, due to the dragging of the posterior roots caused by the increased brain pressure. These changes may be caused by tumours at any part of the brain, but those situated in the posterior cranial fossa are particularly apt to produce increase of the cerebral pressure and thus to make their influence felt upon the spinal cord. This view is supported by the experiments of Finkelnburg (*A. f. kl. M.*, Bd. lxxvi.). Nageotte (*R. n.*, 1904) and Raymond (*Arch. de Neurol.*, 1904, and *R. n.*, 1906) have carefully studied this question. According to their investigations, the congestion of fluid extends chiefly to the periganglionic subarachnoid space of the spinal ganglia, where it produces tension and traction, which affects the ganglion itself. Raymond reports that he has seen it swollen to three times its ordinary size. See also the work of Duret (*loc. cit.*), Margulies (*R. n.*, 1907), and Lejonne ("L'Encéphale,"

1907). Some writers (Dinkler, Becker) think that toxic influences and disorders of nutrition are also concerned, a view which finds some support in the results of experimental lumbar anæsthesia.

Numerous changes of the nervous elements of the brain are also found in cerebral tumour, and these have been attributed to toxic effects arising from the tumour (Dupré, Devaux, E. Müller, Weber-Papadaki; see also the Leber-Deutschmann theory of choked disc, p. 715). An attempt has been made to explain the auditory disturbances by the assumption of a similar injury to the acoustic nerve from lymph stasis in the inner ear (Souques, *R. n.*, 1904). Fuchs (*Z. f. H.*, xxiii.) speaks of tertiary degenerations in cerebral tumour, among which he includes those which develop at distant sites, *e.g.* in the posterior roots.

Symptoms.—Brain tumours give rise to a number of symptoms which are independent of their localisation, viz. *general brain symptoms.* Other symptoms are due to lesion of certain parts of the brain; they vary with the site of the neoplasm and are known as *focal* (or *localising*) *symptoms.*

Among the former we include *choked disc, headache, stupor, vomiting, vertigo, slow pulse,* and *general convulsions.*

Of these the more important are the symptoms which can be objectively recognised. The most important of all is optic neuritis or choked disc. Choked disc is so characteristic of brain tumour that it is due in 90 out of 100 cases to this cause, and is entirely absent only in small number of cases (10 to 20 per cent.). It is almost always bilateral, although it is often more marked in one eye than in the other, usually in the eye of the affected side, but also often in the opposite one. Gunn's view that the eye of the same side is affected in tumour of the frontal lobes, and that in tumour of the occipital lobes the opposite eye is more early and markedly involved is by no means universally applicable. Optic neuritis is nearly allied to choked disc, and is indeed merely choked disc in a lesser degree. It often precedes the development of a true choked disc, and is in itself less pathognomonic than the latter, as slight degrees of optic neuritis may occur in various morbid processes.

Headache is an almost constant symptom of cerebral tumour. It may of course be absent in the initial stages, or it may be slight and intermittent. It is, however, very seldom absent throughout, although we should remember the possibility of this occurrence. It is violent to a degree rarely produced by other diseases, and in the later stages of the illness it is persistently present, although it may become at times markedly intensified. It is usually increased by any forcible expiratory movement, *e.g.* coughing, straining, sneezing. It yields neither to mental treatment nor, permanently at least, to the drugs which cure simple (nervous) headache. It is usually a diffuse pain spreading over the whole head, but in many cases it is felt at certain sites, *e.g.* the frontal or occipital regions. We shall later discuss the question as to how far its site may be useful in local diagnosis.

Stupor is a very important symptom. It may, indeed, be absent during the initial stage of the illness when the tumour is very small or when it is situated outside the territory of the cerebrum, and is therefore extra-cerebral, in the base, the cerebellum, etc. It may otherwise be taken to be the rule that the mental functions are always involved at the height of the disease. The patient may certainly be able to answer questions, but evidently with difficulty. He resembles a man *overpowered with sleep*, and in the advanced stages this condition becomes so marked that he may fall asleep while eating, let the food remain in his mouth, and

pass the urine and fæces involuntarily. He may lie for weeks and months as if wrapped in deep sleep. Stupor in its slightest form usually appears at an early stage.

Mental disorders of other kinds are seldom caused by brain tumour. Conditions of melancholy, hallucinatory excitement, simple dementia, delirium, etc., and a form of mental weakness associated with peculiar attempts to be facetious have been observed. We shall have to return to this point when we discuss the question of localisation.

Vomiting is not a constant symptom, but it is present in the majority of cases. It has all the peculiarities of cerebral vomiting, and it may be one of the early symptoms, although it usually only appears long after the headache. It is specially apt to occur when the headache is at its height. It is most marked in tumours of the cranial fossa (of the cerebellum and the oblongata).

Vertigo.—The patient very often complains of a persistent, confused feeling in the head, like that of intoxication. This sensation is known as vertigo. Attacks of true giddiness, in which the patient loses his balance and either falls down or has to hold on to something to prevent himself doing so, are less common. This symptom is usually most severe in tumours of the cerebellum.

Attacks of *loss of consciousness* with *convulsions* occur very frequently in brain tumour. This condition is quite different from the cortical epilepsy, which only appears when the tumour occupies a certain site. Amongst the general symptoms, as they are defined above, we must include attacks of the type of true epilepsy, which occur at any stage of the disease, and may be a prodroma of it. The epileptic attacks may precede the onset of the other symptoms so long (for years and even for decades) that it is doubtful whether the epilepsy should be regarded as an initial symptom. It should be rather considered as an independent disease and as suggesting the possibility that a brain thus organised is specially predisposed to the development of a neoplasm. Thus cases have been described by Sharkey, Bruns, Oppenheim, Bowlby, Giannelli, Mingazzini, Bindo de Vecchi, Ballet and Armand-Delille, Dide, Creite,[1] and Simmonds,[2] in which the epileptic fits have existed for ten to thirty years before the symptoms of brain tumour became manifest. In several of these cases there was an osteoma, a partially ossified growth (angioma, etc.), or a solitary tubercle, which would make it seem probable that the epilepsy was really the sign of the new growth or possibly (?) of a local inflammatory process which was also the cause of the tumour which developed later. There may, instead of typical epilepsy, be *attacks of simple loss of consciousness*, which again may be associated with *automatic* and *forced movements*; on the other hand there may be simple convulsions, consciousness being retained or but slightly impaired. The spasms may bear a great resemblance to hysterical attacks. Successive attacks of tetanic contraction of the muscles of the body with retraction of the head, in which the patient is quite conscious or but slightly confused, have often been observed in tumours of the posterior cranial fossa (Jackson, Horsley [3]).

In a great number of cases the *pulse rate falls* to 48 beats per minute. or even less; this condition may be persistent, but is usually transitory. This is by no means an early symptom. It is a sign of the increased

[1] *M. m. W.*, 1903. [2] *N. C.*, 1905. [3] *Br.*, 1907. See the Bibliography here.

intracranial pressure, and is as a rule preceded by other symptoms of this condition. It may of course develop in tumours which directly involve the vagus, but in such cases the symptom of vagus paralysis, *i.e.* the acceleration of the pulse, which in other cases of cerebral tumour we meet with in the terminal stages, soon tends to replace the symptom of vagus irritation, *i.e.* retardation of the pulse.

Focal (or *Localising*) *Symptoms.*—There are cases of brain tumour in which the general cerebral symptoms or some of these alone are present during the whole course of the illness. In such cases we can merely give a diagnosis of brain tumour, as we are not in a position to form any correct idea of its site. If we except the early stages of the disease, the number of these cases has, it is true, gradually diminished of late years. Small tumours run their course at almost any site without giving rise to localising symptoms, especially in the white matter, where they simply compress the fibres without impairing their power of conduction. Large tumours do not necessarily give rise to characteristic localising symptoms if they are situated in the right frontal or in the right temporal lobes. It would seem that there may be more or less complete destruction or loss of function of these portions of the brain without the inevitable appearance of any marked symptoms. At any rate we are not sufficiently familiar with such symptoms to recognise and use them for the purpose of diagnosis. There was a time when it was believed that no attempt should be made to *localise* the tumour. This belief was based on the reasons given above, and also on the more important ground that new growths may have an irritating or a paralysing effect upon portions of the brain which are widely removed from their seat. These symptoms were known as "*distant-effects*"—in opposition to *focal* or *neighbouring symptoms* (Bruns)—and their significance was certainly over-estimated. It is conceivable that the pressure exercised by a tumour, even though it be felt throughout the whole of the brain, may affect to a greater degree the parts adjacent to it than the parts at a distance, that it may have a more marked influence upon the tissue of the corresponding than upon that of the opposite hemisphere, etc. etc. It is obvious, therefore, that the focal symptoms must lose something of their localising value when the symptoms of general brain pressure are very severe, and especially when the tumour is accompanied by marked hydrocephalus or swelling of the brain (Reichardt). Localising focal symptoms may also be absent when the tumour is of very slow growth. Thus in one case (Remak-Seiffer) a basal tumour of fifteen years' standing, which had caused severe compression and displacement of the cerebellum, had given rise to no focal symptoms pointing to its seat of origin. Edinger has given us a very interesting report of a similar case. Nevertheless, the results of brain surgery during the last twenty to thirty years afford striking proof that focal symptoms are of immense value in the recognition and localisation of tumours of the brain.

Tumours of the Motor Zones.—Tumours of this kind give rise to the most pronounced focal symptoms, as they very often appear a considerable time in advance of the general cerebral symptoms. Symptoms of cortical epilepsy constitute, as a rule, the first manifestation of the disease. A tonic spasm of the muscles occurs, or convulsions, beginning in one extremity, the face, or in one group of muscles, limited at first to these, but spreading later, or in subsequent attacks, in a regular way

over the whole of the affected side of the body. Consciousness is retained, if not throughout the attack, at least during its commencement (see pp. 677 *et seq.*). These symptoms of irritation are followed by those of paralysis, which may at first be transient in nature, appearing only at the end of the attack, but which gradually increase in intensity and extent until they finally become constant. They have almost always the character of *monoplegia*—brachial, crural, facial, or facio-brachial, etc. etc., according to the site of the tumour. The dissociated appearance of the paralysis is specially characteristic of new growths in the motor area. As the tumour increases in size, the monoplegia may develop into hemiplegia. The convulsive attacks may be ushered in by paræsthesiæ in one extremity or in the face, or these may in themselves represent the attack, but they are usually accompanied or soon followed by the convulsions. There is frequently also at the site of the paræsthesiæ some objective diminution of sensation, particularly of the tactile sense and the sense of position (see p. 627).

Attacks such as those described justify the opinion that the tumour is situated in the cortex of the motor zone or in its immediate neighbourhood (meninges, subcortical white substance, adjacent areas of the frontal lobes, etc.). The onset of the twitching in a certain muscle and the subsequent paralysis of this muscle show that its centre is the seat of origin of the tumour. This is really the way by which the centres of the various muscles have been localised in the brain. It is naturally not to be expected, in view of the irregular extension of many tumours and of the different directions in which they grow, that all these symptoms will develop with the regularity of an experiment. The attempt, which has again lately been made by Valkenburg (*N. C.*, 1906), to distinguish between cortical and subcortical tumours has not yet furnished results which can be depended on. It should further be remembered that a hæmorrhage occurring within a tumour may cause instantaneous and widespread paralysis. The tumour may remain latent for a long time, and may, as the result of a hæmorrhage within it or of a softening in its neighbourhood, suddenly give rise to symptoms of paralysis not preceded by any symptoms of irritation. Attacks of paralysis of short duration may apparently occur instead of the attack of cortical epilepsy (Higier, Binswanger). Further, a new growth of the motor region may produce convulsions which differ from the type of cortical and resemble entirely that of genuine epilepsy. All these possibilities must be kept in mind. It must also be remembered that the areas of the frontal and parietal lobes adjacent to the central convolutions may also be the seat of the tumour, which may only approach the motor region sufficiently near to produce corresponding symptoms. Tumours of the central ganglia may also give rise to symptoms of motor irritation and inhibition of this kind, but these only develop during the later course of the disease.

The symptoms here described are chiefly produced by glioma (as in the case illustrated by Fig. 334), sarcoma, syphiloma, tubercle, and cysticercus.

As regards the conditions of the muscles of the trunk, pharynx, jaw, larynx, and forehead during cortical epilepsy, and the *sensory* symptoms of irritation and paralysis corresponding to the motor symptoms, the reader should consult p. 678. We would also refer

to the various sections with reference to the state of the tendon reflexes, of the tonus, and nutrition of the muscles in cortical paralysis.

If the new growth is seated in the paracentral lobe, corresponding symptoms may develop early in both legs or in both sides of the body. I have been able to report several cases of this kind, and Souques (*R. n.*, 1906) has described a striking example.

Vasomotor disturbances and tachycardia have only been observed in rare cases of tumours of the motor region.

Tumours of the Speech Centre.—The tumour not infrequently occupies

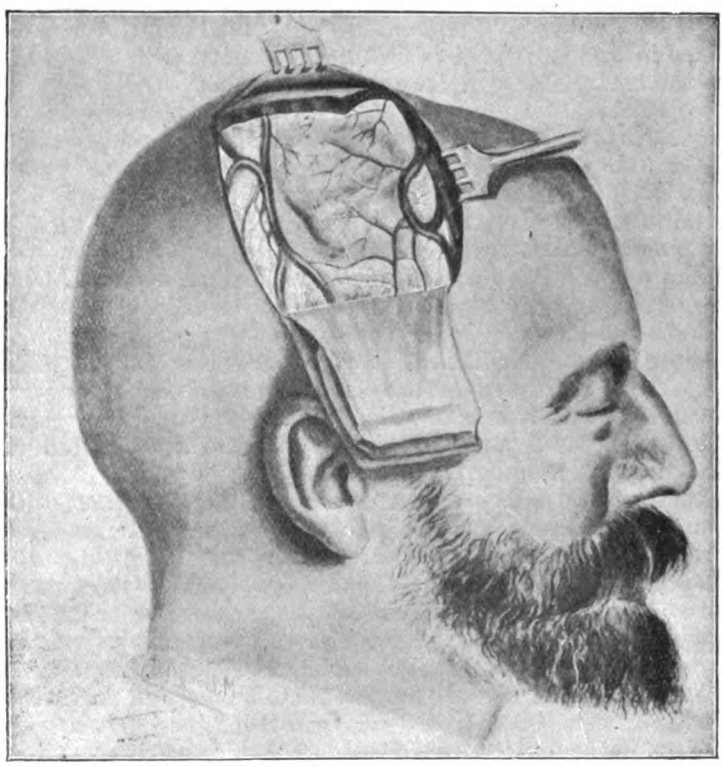

FIG. 334.—Cerebral glioma of central region. Drawn after surgical exposure. (Oppenheim-Krause.)

a site in the left frontal or temporal lobe. If the third left frontal convolution or *Broca's* area be itself affected, motor aphasia as a rule develops early, and usually slowly. The disorder is slight at first, and is only noticeable at times, but it gradually becomes more and more complete and permanent. If the tumour is seated in other parts of the frontal lobe, the speech may be for a long time or entirely unaffected. As the tumour grows, the function of the speech centre usually becomes more and more impaired. I have been struck with the fact that at first there is generally a kind of disinclination to speak, or dumbness, although the patient is still quite able to name every object and to find all the words whenever he is obliged to talk. But he speaks slowly; his answers come not only languidly, but sometimes with the symptoms of marked

bradylalia. Under these conditions a slight dysarthria may precede the development of the aphasia or may be combined with it. Aphasia may be entirely absent in cases of growths arising from the orbital surface of the left frontal lobe. Thus I have seen aphasia occur only towards the close of life in a case of tumour which, arising from the dura, had penetrated deeply into the underlying surface of the left frontal lobe. I have found this also in the case of a left-handed person where a growth of the size of a fist, which had severely compressed the frontal lobe in an inward and upward direction, had caused no trace whatever of aphasia. The aphasia has, however, been absent even in some cases (described specially by Bramwell) in which the tumour had its seat in the motor speech centre itself—in Broca's area. In the case of a child of four which is illustrated by Fig. 345, there was no sign of aphasia

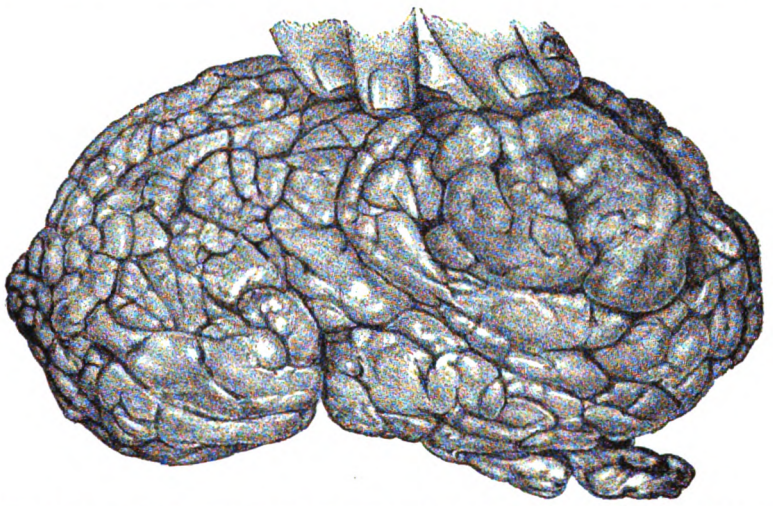

FIG. 335.—Tumour of left supra-marginal gyrus with slight involvement of the first temporal convolution. (Oppenheim.)

until the end of life. The cases described by Ballet and Armand-Delille are interesting in this respect.

Tumours of the left temporal lobe, in particular those affecting the first convolution, give rise to word-deafness, amnesic aphasia, and paraphasia. In one case under my observation [1] the tumour which arose from the supramarginal gyrus lay in the posterior area of the Sylvian fissure and pressed upon the first temporal convolution (Fig. 335), penetrating into it to some extent. In this case the aphasia only became apparent when the patient sat upright.

Tumours of the temporal lobe [2] may occasionally cause convulsions or disturbances of consciousness which are ushered in by an auditory aura—tingling or whistling in the ear of the opposite side (Gowers, Westphal, Oppenheim, Bennet, Wilson, etc.). If the tumour penetrates

[1] *D. m. W.*, 1898.

[2] See the monograph by Knapp: "Die Geschwülste des rechten und linken Schläfenlappens," Wiesbaden, 1905. Also Oppenheim, in "Beitr. z. Diagnostik," etc., Berlin, 1907; Buzzard (*Lancet*, 1906), and Niessl-Mayendorf, etc.

deeply, it may give rise to hemianæsthesia, hemianopsia, and hemiplegia, due to involvement of the corresponding nerve tracts.

Hemianopsia may be produced by pressure on the optic tract, and tumours in the median aspect of the temporal lobe may also cause focal symptoms by compression of the cerebral peduncle, as in a case described by Ménétrier-Bloch (*R. n.*, 1906).

The symptoms of growths from the temporal lobe may include *disturbances of smell* and taste, hallucinations of this kind, attacks of confusion and impairment of consciousness (with or without convulsions), which are preceded with corresponding sensory disorders (Jackson, Clarke, Pitt, Linde, Siebert), anosmia, or hyposmia. These symptoms point to the fact that the tumour has either originated in the median areas of the temporal lobe, the gyrus fornicatus, and the uncus, as I [1] have been able to demonstrate in one case which I have described, or has involved these regions. We must, however, remember that direct lesion of the olfactory nerve may also be the cause of these symptoms. Further, a few rare cases have been observed in which a large tumour in the temporal lobe (Bartels) and even total extirpation of this lobe (Haidenhain) have failed to affect the sense of smell.

A neoplasm situated deep down in the white matter, in the *hippocampus major*, and even in the *central ganglia*, may, if it is large enough and if its growth is in the direction of the speech centre or of the tracts coming from it, give rise to aphasia, usually of a mixed character and incompletely developed. An impure form of aphasia may also occur in tumours of the left island of Reil.

I may remark parenthetically, that I have observed glycosuria in several cases and reflex immobility of pupils in two cases of tumour of the temporal lobe. The latter has also been found by Knapp and by Rosenblath (*Z. f. N.*, xxxi.). It was probably merely the symptom of a distant effect, the significance of which it is difficult to determine.

Tumours which involve the region of the *left inferior parietal lobe*, either directly or by means of pressure, may give rise to *alexia* and agraphia. If they occupy the basal posterior part of the left parietal and the adjacent parts of the occipital lobes, a form of aphasia may develop which we have already described as *optic* aphasia.

In left-handed individuals, tumours of the corresponding parts in the right hemisphere of the brain may produce the various forms of aphasia. This has occasionally been found to occur in right-handed persons also (see p. 736).

Apart from aphasia, we know of no characteristic localising symptom of tumour of the *frontal lobe*, but a number of signs have acquired a certain value in respect of the local diagnosis. Thus it would seem that psychical disorders (simple dementia, facetiousness [2]) tend to develop early and to be very marked in such cases. In one case of this kind which I have observed along with Bergmann, this symptom was most marked and it disappeared entirely within a short time after the removal of the tumour. Devic-Courmont and Friedrich have also seen recovery from the psychosis after removal of the tumour. Cases of tumour of the frontal lobe have also been observed, however, in which there was

[1] *Mitt. aus d. Grenzgeb.*, vi.

[2] I have given the name "Witzelsucht" to this mental symptom (*A. f. P.*, xxi.), which Jastrowitz had formerly termed "moria."

no mental disorder of any kind. In any case, it has been proved that the mental powers may be severely impaired in cases of tumour affecting any other region of the brain.

The question has been much discussed by Bernhardt, Jastrowitz, Oppenheim, Bruns, Anton-Zingerle, Durante, Gianelli, Cestan-Lejonne, and lately with special care by Schuster ("Psych. Störung. bei Hirntumoren," Stuttgart, 1902), and E. Müller (*Z. f. N.*, xxii.). The latter strongly contests the relation between the frontal lobe and the mental functions, and attributes the mental disorders rather to the general increase of brain pressure, which is apt to be specially marked on account of the size usually attained by growths in the frontal lobes. Reichardt also maintains that the mental disturbances are due to the general affection of the brain (pressure, swelling, or atrophy). See also Knapp, *Br.*, 1906.

Tumours of the posterior region of the frontal lobe may give rise to convulsions by involving the motor zones. The first symptom of such a spasm may be conjugate deviation, which points to its frontal origin, but this is not a definite focal symptom. Grainger-Stewart [1] lays special weight on the early disappearance of the abdominal reflex on the opposite side (?), and on the fine tremor of the same side. Rigidity of the neck and the so-called cerebellar ataxia also occur in tumours of the frontal lobe. Bruns, who was the first to emphasise the existence of this frontal ataxia, is of opinion that it is specially apt to occur in tumours of the marginal gyrus, which involve the corresponding areas of both frontal lobes.

Tumours arising from the *parietal lobes* may produce symptoms of motor irritation and inhibition (from compression of the motor zone). Disturbances of sensibility, especially of the sense of position and of stereognostic perception, and ataxia have been found in tumours of this region by Oppenheim,[2] Mills, Spiller, and others (the so-called mind-paralysis being less common, see p. 681). Alexia and optic aphasia may be symptoms of growths arising from the left inferior parietal lobe. If the tumour penetrates deep into the substance of the inferior parietal lobe, it may give rise to hemianopsia.

Tumours of the Visual Region.—Tumours of the optic tract, of the posterior tubercle of the optic thalamus, of the region of the corpora quadrigemina or the external geniculate body, as well as those which interrupt the optic radiations on their way to the occipital lobes, and those which are situated in the occipital lobe itself, all give rise to the same focal symptom, viz., *hemianopsia*. This symptom would therefore be of no great significance as regards local diagnosis if the accessory symptoms did not give some indication as to the seat of the lesion. Tumours of the optic tract usually involve other basal cranial nerves, especially the oculo-motor nerve. Tumours of the optic thalamus lead as a rule to an affection of the motor, and especially of the sensory tract. If the corpora quadrigemina are involved, there is usually oculo-motor paralysis, etc. If the optic tract is affected on its way to the left occipital lobe, aphasia may be combined with the affection of sight. Finally, should the tumour attack the occipital lobe itself, hemianopsia is the only direct focal symptom. In a few cases unilateral hallucinations of vision also appeared. We have had an opportunity of proving that both the hemianopsia and the visual hallucinations are cured by successful removal of a tumour of the occipital lobe (Fig. 336). In many cases of new growths

[1] *Lancet*, 1906; *R. of N.*, 1906. [2] *A. f. P.*, xxi; *Mitt. aus Grenzgeb.*, vi.; *M. f. P.*, xviii.

in this region, however, there are no focal symptoms whatever. Bruns concludes from his own experience that choked disc is comparatively rare in tumours of the occipital lobe, but he states that he has seen transient blindness in several instances. Bilateral and indeed unilateral tumours of the occipital lobe may give rise to mind-blindness (Ferrier, Jackson, Gowers, Pooley, Wollenberg, Bruns, Bramwell, etc. ; see p. 723).

Rosenblath (*Z. f. N.*, xxxi.) describes cortical blindness from diffuse sacromatosis of the meninges over both occipital lobes.

Tumours in the Grey Matter of the Cerebral Hemispheres and in the Central Ganglia.—The diagnosis of such cases is often a very difficult matter,

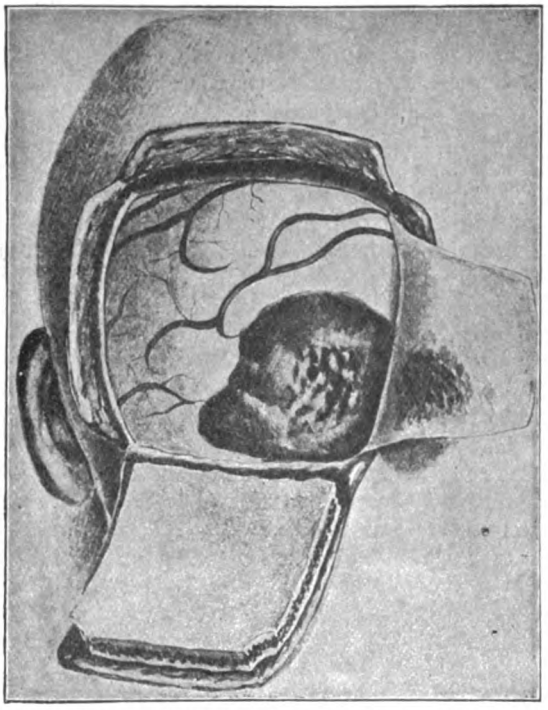

FIG. 336.—Tumour of the left occipital lobe. Drawn during operation. (Oppenheim-Krause.)

but it is also one of little practical interest. The focal symptoms may be absent for a long time, or even altogether. But as the internal capsule is usually affected, either directly or from the pressure, symptoms of *hemiplegia* are apt to appear. These are not as a rule sudden in their onset, but develop gradually and incompletely. According to my experience the " distant effects " of the lesion on the motor tract first become apparent in the facial nerve and in the changes of the reflex excitability which correspond to those of a pyramidal lesion. Symptoms of motor irritation may also appear in the form of unilateral spasms of indefinite character, which, however, do not correspond exactly to the type of cortical epilepsy, and also of hemichorea, hemiathetosis, tremor, forced movements, and automatic movements. In cases of new growths

which injure the pyramidal tract directly or through pressure, the hemiplegia is usually associated with spastic symptoms, the tendon reflexes being exaggerated and Babinski's sign, etc., being present. The general increase of the cerebral pressure or the lesion of the posterior roots to which it gives rise, may, however, have a compensating effect, so that the tendon reflexes are diminished or abolished. They may also be absent only on the unaffected side (Oppenheim), and under these conditions there is a difference between the response of the knee and Achilles jerks. It is not unusual, in my experience, to find that the Achilles jerk is present or exaggerated although the knee-jerk is absent. Hemianæsthesia and hemianopsia of the opposite side are also common symptoms. Tumour of the optic thalamus may also cause paralysis of the movements of expression of the facial nerve (see p. 651), forced laughter, and less frequently loss of the automatic movements of the facial muscles.

The greater the pressure of the tumour upon the cortex, either from the first or on account of its subsequent growth, the greater is the tendency to the development of corresponding focal cortical symptoms, due to the pressure or distant effect.

Bilateral tumours are comparatively common in the central ganglia, especially in the optic thalamus. The symptoms in such cases are closely allied to those caused by tumours of the brain stem and cerebellum (Sepilli-Lui, Jacobsohn).

Tumours of the corpus callosum do not as a rule cause very definite symptoms. Bristowe notes the following indications: 1. Gradual aggravation of the symptoms; 2. Absence or insignificance of the general symptoms of tumour (?); 3. Marked impairment of the intelligence, hebetude, somnolence, and a non-aphasic disturbance of speech; 4. Hemiparetic symptoms, which are often associated with less marked paresis of the other side of the body, one side being usually affected before the other; 5. Absence of any symptoms of involvement of the cranial nerves.

Bruns and Giese, whose observations have been supplemented by those of Devic-Paviot, Beevor, Zingerle, Blackwood, Greenleess, D'Allocco, Starr, Berkley, Brissaud, Würth, Putnam-Williams (*Journ. Nerv. and Ment. Dis.*, 1901), Steinert (*Z. f. N.*, xxiv.), Bregmann (*Z. f. N.*, xxix.), and others, have found similar conditions, but regard them as very indefinite diagnostic signs. A patient of Touche's showed symptoms which differed essentially from those just described. Schupfer has endeavoured to establish certain criteria from the localisation of tumours within the various sections of the corpus callosum, but his data have been contested. Mingazzini (*M. f. P.*, xix.) found in one case that the general symptoms were very marked, whilst a bilateral hemiparesis was the only focal symptom. Raymond (*R. n.*, 1906) regards the mental disturbances as the only characteristic signs, and he tries to describe their peculiarities. Zingerle speaks of a "corpus callosum ataxia," in view of the fact that his patient lost the power of walking, but the stuporous imbecility of the patient made it difficult to judge of this condition. In his case, as in a few others, the tumour remained latent for a long time, and then the symptoms appeared in a more or less acute manner, the disease having thereafter a comparatively rapid course. Forster has lately published a contribution to this subject. See also Lippmann, *A. f. P.*, Bd. xliii.; Raymond-Lejonne-Lhermitte, "L'Encéphale," 1906.

With regard to the form of apraxia produced by lesion of the corpus callosum, see p. 779.

Tumours of the region of the corpora quadrigemina are characterised by the following symptoms: oculo-motor paralysis, especially paralysis of the corresponding ocular muscles on the two sides or paralysis of conjugate deviation for upward and downward movements, symptoms of paralysis in the pupils, inco-ordination in standing and walking, deafness, and probably also amblyopia (which is due to injury of the lateral geniculate body). In a case described by Oordt the motor disturbance showed a mixture of ataxia, intention tremor, and athetosis.

[illegible] perform simple move- [illegible] side of the body, but [illegible] tabetic ataxia. There [illegible] A homolateral [illegible] hemisphere and [illegible] combined with hypotonia. [illegible] cerebellum, especially the pyramids, [illegible] same side, as Mann, [illegible] Luciani. The [illegible] is an almost constant, [illegible] cerebellum. In [illegible] throughout the

[illegible] important symptoms which are due [illegible] cerebellum, presses downwards [illegible] *medulla oblongata*, and injures the *cranial* [illegible] situated at the base of one [illegible] anterior portion, the symptoms of irrita- [illegible] upon the trigeminal, facial, auditory, [illegible] manifestation of the disease Thus [illegible] one half of the face, followed by [illegible] paresis of the facial nerve (with [illegible] auditory nerve, etc. These [illegible] characteristic of tumours of the cere- [illegible] and in a number of cases that absence [illegible] under these conditions, be the first sign of [illegible]

[illegible] in the [illegible] discussion [illegible] symptoms of [illegible] absence of the [illegible] Bassoe-Sky- [illegible] Unger [illegible] Siemerling [illegible] stances, [illegible]

Disturbances [illegible] laryngeal [illegible] it may, [illegible] are due to [illegible] affection of the [illegible] in general to [illegible] 332, 333, and [illegible] diagnosis, chiefly [illegible] The tumours [illegible] angle, the special [illegible] nerves, such as [illegible] paresis the paralysis. [illegible] associated with paresis [illegible] cases of tumour of the [illegible] during the last

months of life persistent clonic contractions of the soft palate, the larynx, the vocal cords, etc., occurred. A somewhat similar condition has been reported by Bastianelli and by E. Meyer and Sinnhuber (*B. k. W.*, 1907). In several instances choreic tremors, or a motor disorder greatly resembling hemichorea, were among the symptoms of tumour of the cerebellum and of the superior cerebellar peduncle. Compression of the pons may give rise to *paralysis of conjugate deviation* towards the side of the tumour. Alternating hemiplegia may also have this origin. Nystagmus is a common and very valuable sign in cerebellar tumour. Many writers regard it as a direct focal symptom. I myself have very rarely seen the Magendie skew deviation of the eyes (see p. 701), to which Stewart and Holmes also refer, and then only after surgical operations on the cerebellum. It has been attributed to lesion of the middle cerebellar peduncle. It is obvious that pressure upon the medulla oblongata may lead to respiratory troubles, but when these appear the end is usually not far off.

If the compression acts upon the pyramidal tract, hemiparesis or paraparesis occur. The hemiparesis is sometimes contralateral, sometimes homolateral, according as the pyramid of the same or of the opposite side is most affected. The paralysis is then spastic, if the general increase in the brain pressure does not have a compensating effect (see above).

It is a very interesting fact that symptoms of tumour, in general, but of the posterior cranial fossa in particular, are often influenced by the *attitude of the head*, or the *position of the body*, so that the patient carries his head always in a certain attitude, although there is no contracture or paralysis to cause him to do so. Careful examination often reveals the fact that the headache, vomiting, vertigo, etc., may be intensified, or brought on, by changes in the position of the head. We find, therefore, that the patient assumes a certain position when he lies down. If he is compelled to alter it, severe troubles may ensue. I [1] have already pointed out the great importance of this symptom, which has hitherto been neglected in the consideration of so-called forced-positions and attitudes. Hallopeau-Girandeau, Schulz, Pichler, and Schmidt have reported valuable cases of this kind. The last named has proved in several cases that in tumours of one side of the cerebellum (the left, for instance), the opposite lateral position (the right) is avoided, because the patient instinctively tries to prevent the pressure upon the Sylvian aqueduct or the vena magna Galeni which would be caused by displacement of the tumour. I have found in two cases that nystagmus, accompanied by feelings of discomfort, only occurred when the patient assumed a certain lateral position. I have not been able to discover any regularity in these relations, in Schmidt's sense, but I agree with him that the symptom in itself points to the probability of the tumour being situated in the posterior cranial fossa. Finkelnburg has, it is true, seen a similar condition in a case of tumour of the corpus striatum. From my own experience I can merely conclude that the patient tries to assume the attitude of his head, and in lying down the position of his body in which he actually feels least discomfort. These conditions may alter in the course of the disease (from a change in the direction of the growth, etc.). Batten describes as more or less constant, an attitude of the head in which the chin is turned towards the side in which the tumour [illegible]ated, the

[1] *D. m. W.*, 1898, and *Z. f. prakt. Ärzte*, 1900.

Nystagmus is a common symptom. In some cases, however, it only becomes evident when the patient looks in certain directions or during the movement of convergence. Vasomotor disorders (cyanosis of the extremities, rise of the temperature of one side of the body, etc.) have occasionally been noted in tumours of this region (Bruns, Collins, Heubner, Sorgo, Gordinier). Neoplasms of the *pineal gland* give rise, as a whole, to similar symptoms. They may also cause trochlear paralysis and nystagmus. The general symptoms are usually very marked. In a case described by Slawyk and Oestreich [1] excessive development of the penis and mammæ were also symptoms of the disease. Henrot has described a similar condition. Biancone [2] mentions exaggeration of the sexual impulse and priapism. I have seen the genital region completely covered with hair in a girl of six years of age who was suffering from cerebral tumour. This growth had commenced in her second year. Clarke, in one case where a tumour as large as a walnut occupied the medullary velum and the corpus quadrigeminum, found neither ophthalmoplegia nor cerebellar ataxia. Thus tumours in the various sections of the mid and hind brain may run their course without giving rise to any localising symptoms.

As regards the literature upon tumours of the region of the corpus quadrigeminus, we should advise reference to Bach, *Z. f. Aug.*, viii.; Marburg, *W. kl. W.*, 1905; and Uhthoff, Graefe-Saemisch "Handbuch," 2nd edition, Bd. xi., chap. xxii., in addition to the works already mentioned.

The focal symptom of tumour of the *cerebral peduncle* is hemiplegia alternans superior, *i.e.* crossed paralysis of the oculomotor nerve and the extremities. In tumours of this region an intention tremor, and a tremor of the type of paralysis agitans (Gowers, Henoch, Mendel, Blocq-Marinesco, Raymond-Cestan, Oppenheim, etc.), have often been noted. The combination of this tremor with crossed oculo-motor paralysis has, on Charcot's [3] suggestion, been termed by French writers Benedikt's syndrome (see p. 697). In a case described by Sorgo the tremor developed and spread in a peculiar dissociated way. The combination of oculo-motor paralysis and hemiataxia of the other side is less common (according to Krafft-Ebing it is due mainly to the localisation of the tumour in the tegmental region, whilst D'Astros-Hawthorn [4] regard this syndrome of Benedikt along with other signs as characteristic of an affection of the tegmentum). The opposite oculo-motor nerve may become affected during the further course. Collins [5] has described tumour of the Sylvian aqueduct. The drowsiness and tendency to comatose conditions are said to be specially marked in new growths of the third ventricle (Mott), but I have found this symptom absent in one case. In another described by Soca, in which the tumour arose from the hypophysis, the somnolent condition lasted for seven months.

Tumours of the cerebellum [6] may remain latent, or at least they may produce no focal symptoms (cases of Andral, Nothnagel, Ebstein, Couty,

[1] *V. A.*, Bd. clvii.
[2] *Riv. speriment.*, 1899.
[3] "*La Méd. mod.*," 1893.
[4] *R. n.*, 1902.
[5] *Amer. Journ. Med. Sc.*, 1895. For further literature see Uhthoff, Graefe-Saemisch "Handbuch," 2nd ed., xi.
[6] Recent literature: Stewart and Holmes, *Br.*, 1904 (report of forty carefully observed cases, twenty-two of which came under operation or autopsy); Oppenheim, *loc. cit.*; Duret, *R. n.*, 1903; Ziehen, *Med. Klinik*, 1905; Borchardt and Seiffer, with discussion, *N. C.*, 1905; Seiffer, "Beiträge zur Med. Klinik," 1907; Mills-Frazier and Weisenburg, "Tumours of the Cerebellum," New York, 1905. See further on the literature on new growths of the cerebello-pontine angle.

Taylor, Wadsworth, Bramwell, Putnam, Marchand). This, however, is very unusual. They give rise as a rule to a very characteristic clinical picture. The headache is felt specially in the *occipital* region, in the *neck*, and even in the upper part of the back. There may also be frontal headache. There is often a slight degree of *rigidity of the neck*. General and unilateral convulsions, and specially attacks of opisthotonus and tetanic contraction of the muscles of the body (Jackson, Horsley[1]), may occur. Attacks of sudden vertigo with loss of equilibrium, nystagmus, mental confusion, and other accessory symptoms have been described by Dana,[2] Ziehen, and others. Choked disc develops very early, is exceedingly marked, and is always bilateral.[3] There is sometimes great tenderness on percussion of the occipital region. The most important focal symptom, though not a constant one, is *vertigo* and especially *impairment of co-ordination*. These are hardly ever absent in tumours of the vermiform process. The patient rarely complains of persistent vertigo ; it generally comes on in paroxysms, especially when he changes his position, *e.g.* in rising from the recumbent posture.

Stewart and Holmes have tried to characterise this vertigo more precisely in regard to direction, etc., but it does not seem to me that they have succeeded in proving their points. According to their view, the patient has a feeling that external objects are apparently moving from the side of the tumour towards the unaffected side, and that he himself is being pulled in the same direction. This illusory movement of his own body assumes the opposite direction in extra-cerebellar tumours of the posterior cranial fossa.

The disturbance of equilibrium first becomes evident during walking. The patient is apt to fall and staggers from one side to the other, like a drunken man. It is often intensified if the eyes are closed, and the patient may even fall down ; but this is by no means the rule.

I cannot agree with Starr, Beevor, Stewart, and Holmes, that there is any regularity as regards the direction of the swaying. I have only seen very marked swaying and deviation towards the affected side in acute lesions and after surgical operations on the cerebellum. It also appears to follow this direction as a rule in diseases of the vestibular apparatus. Weakness in the muscles of the trunk has sometimes been observed, especially by English writers.

I have seen cases of this kind in which standing and walking were impossible on account of the absolute loss of balance. I have also seen the cerebellar asynergy described by Babinski (p. 28) in a few cases of cerebellar tumour. I can say nothing from my own experience as regards the hemiasynergy in the leg of the same side (combined with unilateral tremor of the arm), which he also mentions. As to the diagnostic value of the adiadokokinesis, *i.e.* retardation of successive movements, *e.g.* of pronation and supination in the non-paretic limbs, which he also describes, we can lay down no definite law, but we may admit that the symptom frequently occurs in cerebellar diseases and that it corresponds as a rule to the side of the tumour. I have found it to be most marked after surgical operations upon the cerebellum. Whilst uncertainty of gait is a very common symptom of cerebellar tumour, ataxia of the

[1] *Br.*, 1907. [2] *New York Med. Journ.*, 1905.

[3] According to the statistics of Martin (*Lancet*, 1897), choked disc is present in 89 per cent. of cases of cerebellar tumour, but is absent in two-thirds of the cases of tumour of the corpus callosum, of the medulla oblongata, and of the pons. These data are practically identical with my own experience, which I had previously reported. Finkelnburg has found no change in the discs in several cases of cerebellar tumour.

extremities, which becomes evident in attempts to perform simple movements, is less frequent. It may be limited to one side of the body, but it may also be so marked in the legs as to recall tabetic ataxia. There is, however, no objective disturbance of sensibility. A homolateral hemiataxia may be caused by tumours of the cerebellar hemisphere and of the inferior cerebellar peduncle. It is usually combined with hypotonia. It seems to me exceedingly doubtful whether tumours of the cerebellum which do not involve the neighbouring parts, especially the pyramids, can give rise to the symptom of hemiparesis of the same side, as Mann, Probst, Duret, and others have stated in agreement with Luciani. The symptom is rather that of hemiataxia. *Vomiting* is an almost constant, or at least a very usual symptom in tumours of the cerebellum. In such cases it is apt to occur very early and to persist throughout the whole course of the disease.

This brings us to a number of important symptoms which are due to the fact the tumour, arising from the cerebellum, presses downwards or forwards upon the *pons and medulla oblongata*, and injures the *cranial nerves* which originate in them. If it is situated at the base of one cerebellar hemisphere and in its anterior portion, the symptoms of irritation and paralysis due to pressure upon the trigeminal, facial, auditory, and other nerves may be the first manifestation of the disease Thus there may be intractable neuralgia in one half of the face, followed by anæsthesia, trophic disorders, etc., paralysis of the facial nerve (with changes of the electrical excitability), of the auditory nerve, etc. These symptoms are, it is true, specially characteristic of tumours of the cerebellar peduncle (q. v.). I have found in a number of cases that absence of the corneal reflex may, under these conditions, be the first sign of involvement of the trigeminus.

My remarks on this subject may be found in a paper by Wollenberg (*A. f. P.*, xxi.), in the first edition of the "Hirngeschwülste," and in the *Mitt. aus d. Grenzgeb.*, vi. See also a discussion on this symptom in the *B. k. W.*, 1904. I regard it as one of the most valuable symptoms of unilateral tumour of the posterior cranial fossa, and attach special weight to pure absence of the reflexes, unaccompanied by disturbances of the sensibility. In one case reported by Bielschowsky-Unger (*A. f. kl. Chir.*, Bd. lxxxi.) this was the only sign of a tumour of the cerebello-pontine angle. Siemerling (*B. k. W.*, 1908) contributes a supplementary case. Bruns, under similar circumstances, found absence of the reflexes in the same side of the nose and palate.

Disturbances in the region of the vagus, paralysis of the laryngeal muscles, etc., also occur according to the site of the tumour. It may, however, be difficult to determine how far these symptoms are due to an involvement of the nerve roots, and how far to an affection of the medulla oblongata. Their limitation to one side points in general to affection of the nerves. In the cases illustrated by Figs. 332, 333, and 340, and many similar ones, I was able to give an exact diagnosis, chiefly from the symptoms of paralysis of the cranial nerves. The tumours were, it is true, situated in the cerebello-pontine angle, the special symptoms of which we shall discuss later.

The signs of irritation in the region of the cranial nerves, such as convulsive tic, should be noted. These may precede the paralysis. The contracture may also from the very first be associated with paresis of the facial nerve (Oppenheim). In one of my cases of tumour of the cerebellum, pressing upon the medulla oblongata, during the last

months of life persistent clonic contractions of the soft palate, the larynx, the vocal cords, etc., occurred. A somewhat similar condition has been reported by Bastianelli and by E. Meyer and Sinnhuber (*B. k. W.*, 1907). In several instances choreic tremors, or a motor disorder greatly resembling hemichorea, were among the symptoms of tumour of the cerebellum and of the superior cerebellar peduncle. Compression of the pons may give rise to *paralysis of conjugate deviation* towards the side of the tumour. Alternating hemiplegia may also have this origin. Nystagmus is a common and very valuable sign in cerebellar tumour. Many writers regard it as a direct focal symptom. I myself have very rarely seen the Magendie skew deviation of the eyes (see p. 701), to which Stewart and Holmes also refer, and then only after surgical operations on the cerebellum. It has been attributed to lesion of the middle cerebellar peduncle. It is obvious that pressure upon the medulla oblongata may lead to respiratory troubles, but when these appear the end is usually not far off.

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It is a very interesting fact that symptoms of tumour, in general, but of the posterior cranial fossa in particular, are often influenced by the *attitude of the head*, or the *position of the body*, so that the patient carries his head always in a certain attitude, although there is no contracture or paralysis to cause him to do so. Careful examination often reveals the fact that the headache, vomiting, vertigo, etc., may be intensified, or brought on, by changes in the position of the head. We find, therefore, that the patient assumes a certain position when he lies down. If he is compelled to alter it, severe troubles may ensue. I [1] have already pointed out the great importance of this symptom, which has hitherto been neglected in the consideration of so-called forced-positions and attitudes. Hallopeau-Girandeau, Schulz, Pichler, and Schmidt have reported valuable cases of this kind. The last named has proved in several cases that in tumours of one side of the cerebellum (the left, for instance), the opposite lateral position (the right) is avoided, because the patient instinctively tries to prevent the pressure upon the Sylvian aqueduct or the vena magna Galeni which would be caused by displacement of the tumour. I have found in two cases that nystagmus, accompanied by feelings of discomfort, only occurred when the patient assumed a certain lateral position. I have not been able to discover any regularity in these relations, in Schmidt's sense, but I agree with him that the symptom in itself points to the probability of the tumour being situated in the posterior cranial fossa. Finkelnburg has, it is true, seen a similar condition in a case of tumour of the corpus striatum. From my own experience I can merely conclude that the patient tries to assume the attitude of his head, and in lying down the position of his body in which he actually feels least discomfort. These conditions may alter in the course of the disease (from a change in the direction of the growth, etc.). Batten describes as more or less constant, an attitude of the head in which the chin is turned towards the side in which the tumour is seated, the

[1] *D. m. W.*, 1898, and *Z. f. prakt. Ärzte*, 1900.

head being approached to the opposite shoulder. In one of my cases of cerebellar tumour the patient kept his head bent so low that his chin rested upon his breast; if he tried to raise it, vertigo and severe headache at once appeared. Finkelnburg [1] has also described interesting cases of this kind, disturbances of circulation and respiration (Cheyne-Stokes respiration) coming on when the head was bent forwards or backwards. Henneberg and Koch have found similar conditions.

Mott (*Brit. Med. Journ.*, 1904) has seen in a case of tumour of the third ventricle, a marked difference between the condition in the recumbent and in the upright position, and he attributed this to the obstruction thus offered to the flow of fluid to the ventricles.

Finally, we must remember that the hydrocephalus which always accompanies cerebellar tumour may give rise to symptoms which are easily misconstrued. Thus it not infrequently happens in severe hydrocephalus that the floor of the third ventricle, inflated like a bladder, compresses the chiasma and thus gives rise to complete blindness. I have also seen hypophyseal symptoms originate in this way (see below), and further I have repeatedly found *anosmia* (unilateral or bilateral) in cerebellar tumours, which was due to the fact that the olfactory nerve was severely flattened by the pressure exerted upon it by the base of the brain. Muskens has stated that a change corresponding to choked disc may occur in this nerve. In one of my cases a cerebellar tumour had pressed so severely upon the corresponding cerebral hemisphere from below that by kinking the inferior or posterior horn it had produced a hydrocephalus limited to the corresponding posterior horn. It is possible that the local symptoms of lesion of the occipital lobe may also be produced in this way by growths in the cerebellum, and that *vice versa*, growths in the occipital region may give rise to focal cerebellar symptoms by exerting pressure upon the cerebellum. It is a remarkable fact that as a rule consciousness usually remains for a long time unaffected in tumours of the cerebellum. Finally, we should remember that the knee-jerks may occasionally disappear in cerebellar tumour. In such cases there may be a combined disease of the brain and spinal cord (tabes, sarcomatosis, gliosis, etc.). The symptom has often been found, however, in uncomplicated cases of cerebellar tumour (less often in tumours of other regions).

Cases of this kind have been described by Mackenzie, Gowers, Dercum, Pitt, Mendel, Oppenheim, Handfort, Batten and Collier, Peterson, Hawthorne, Voss, Sommer, Raymond, Lejonne, and others. With regard to the cause of this symptom, see p. 893. Brain tumour may also cause a combination of hypotonia with loss of the tendon reflexes and Babinski's sign, should it on the one hand effect the pyramidal tract and on the other cause considerable increase of brain pressure. This has been observed both by Finkelnburg and myself. In one such case I was able to elicit ankle clonus immediately after the dura was opened, whilst the knee-jerk was absent and Babinski sign persisted. Goldscheider and Koch (*Charité-Ann.*, xxviii.) observed return of the knee-jerk after lumbar puncture. Raymond and Lejonne have shown (*R. n.*, 1906) that tumour, by giving rise to congestion of the fluid and degeneration of the roots, may produce a clinical condition like that of tabes.

Tumours of the Pons and Medulla Oblongata.[2]—Tubercle and glioma

[1] *Z. f. N.*, xxi.

[2] Comprehensive papers upon tumours of the pons and of the medulla oblongata have, in addition to those mentioned in the introduction, been published by Delbanco, Zahn, Cymbal (*V. A.*, Bd. clxvi.), Schöler (*Kl. M. f. Aug.*, 1902), Brüning (*Jahrb. f Kind.*, Bd. lv.), Henneberg (*Charité-Ann.*, xxviii.), and Stern (*Z. f. N.*, xxxiv.). For references to the literature, see specially Uhthoff, Graefe-Saemisch's "Handbuch," 2nd edition, Teil II. Bd. xi.

are specially apt to develop in this region (see Figs. 337-339). Tubercle may remain latent for a long time.

Pontine tumours as a rule give rise to symptoms, the focal being more marked than the general. Choked disc is comparatively often absent (Oppenheim, Martin); in my experience, this may at least be taken to be the rule. I would emphasise this more strongly, for instance, than Uhthoff does in his work. The typical focal symptom is *hemiplegia alternans*, the facial, abducens, and trigeminus, or one of these nerves, being paralysed on the one side, and the extremities on the other. There may also be associated oculo-motor paralysis on the side of the tumour. Spitzer describes conjugate deviation of the eyes and head towards the opposite side from the tumour (see also p. 698). In other cases the cranial nerves above named were affected on both sides. Bilateral hemiplegia may also develop, and in such cases dysarthria and dysphagia are almost always present. General, unilateral, or alternating convulsions are less common. Sensory disturbances and hemiataxia have also been

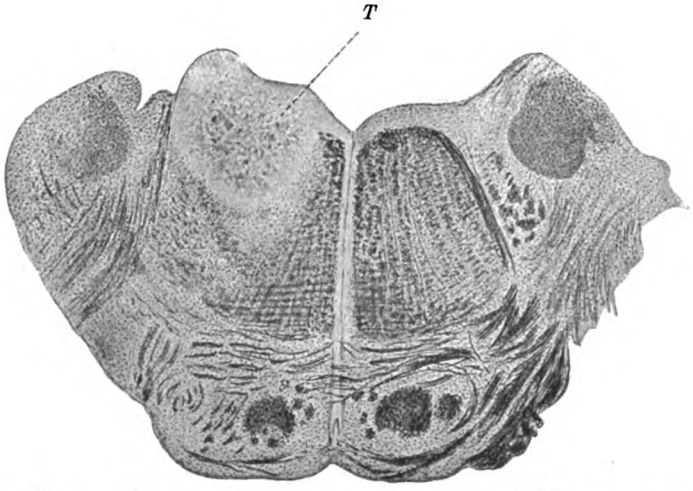

FIG. 337.—Tumour of the pons in the region of the facial and abducens nuclei and their roots (*T*).

observed in pontine tumours, and the paralysis of the cranial nerves may alternate with these symptoms. In one case facial paralysis was the only symptom of a pontine tumour (Hunnius). I have occasionally found that although the motility was conserved in the region of the facial, and although the sensibility in the trigeminal area was normal or but slightly impaired, there was no winking of the eyelid on the affected side, the winking reflex being therefore absent. The auditory nerve may be involved on one or both sides. In several of my cases in which the disease commenced with unilateral facial paralysis and paralysis of the conjugate deviation, the Oppenheim's and Babinski's signs on the opposite leg were present for some weeks before the hemiplegia of the opposite side appeared.

Neoplasms of the *medulla oblongata* may also remain latent, especially when they extend in the direction of the fourth ventricle or develop within it. This has often been observed in cases of cysticercus

(see next chapter). On the other hand they produce symptoms which correspond in many respects to those of pontine tumours, except that they specially involve the eighth to the twelfth cranial nerves. Deafness, deglutition paralysis, dysarthria, aphonia, etc., are among the most prominent symptoms. These are accompanied by irregularity in the action of the heart, respiratory troubles, hiccough, and in many cases by glycosuria, diabetes insipidus, vasomotor symptoms, etc. There may be paralysis of the extremities of one or both sides. Cerebellar

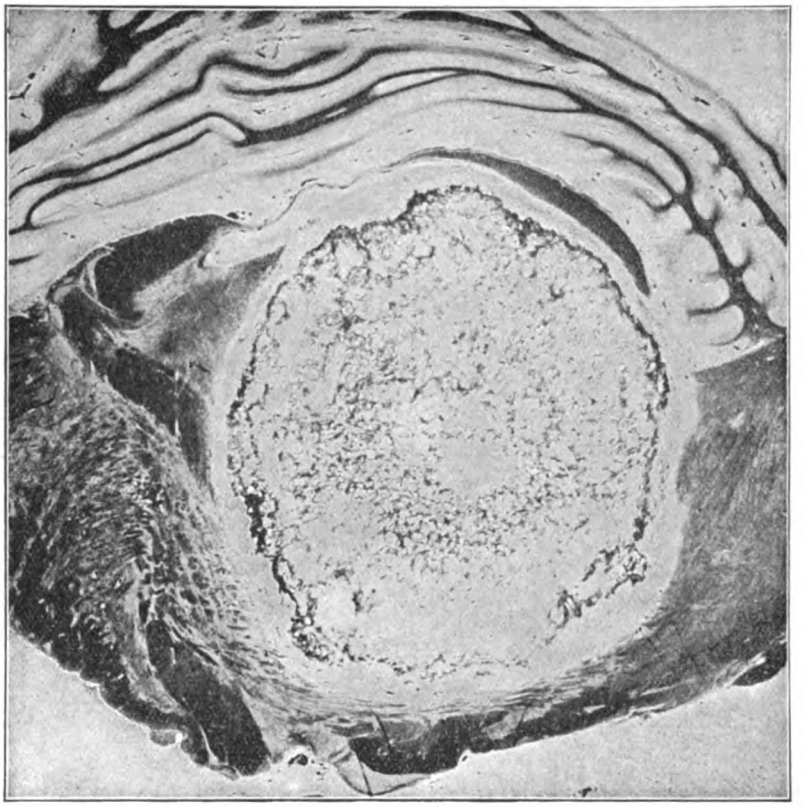

FIG. 338.—Transverse section through a solitary tubercle in the pons. Section in Oppenheim's collection. Pal's stain.

ataxia is common, and motor ataxia of the same or the opposite side may also occur. The general signs of intracranial pressure are often slight. Instead of choked disc there may from the first be atrophy of the optic nerve. We have seen diabetes insipidus and optic atrophy exist for years as the only symptoms.

A. Stern regards occipital headache, forward bending of the head, and an intermittent course as important signs.

Somnolence and mental disorders are special indications that the tumour is seated in the region of the third ventricle. These have in a few cases simulated a condition of paralytic dementia (Mott, Barret,

Henneberg). In a case of cholesteatoma of this region, however, I found that the mental disorders were absent for a long time and that marked stupor and drowsiness appeared only in the later stages. There may in addition be thirst, polyuria, ophthalmoplegia, etc., and the symptoms will vary according as the neighbouring structures, such as the thalamus, corpora quadrigemina, or cerebral peduncle are involved.

Henneberg regards tumours lying free within a ventricle without any connection with the brain tissue, and usually connected with the plexus, and those situated upon the ependyma as

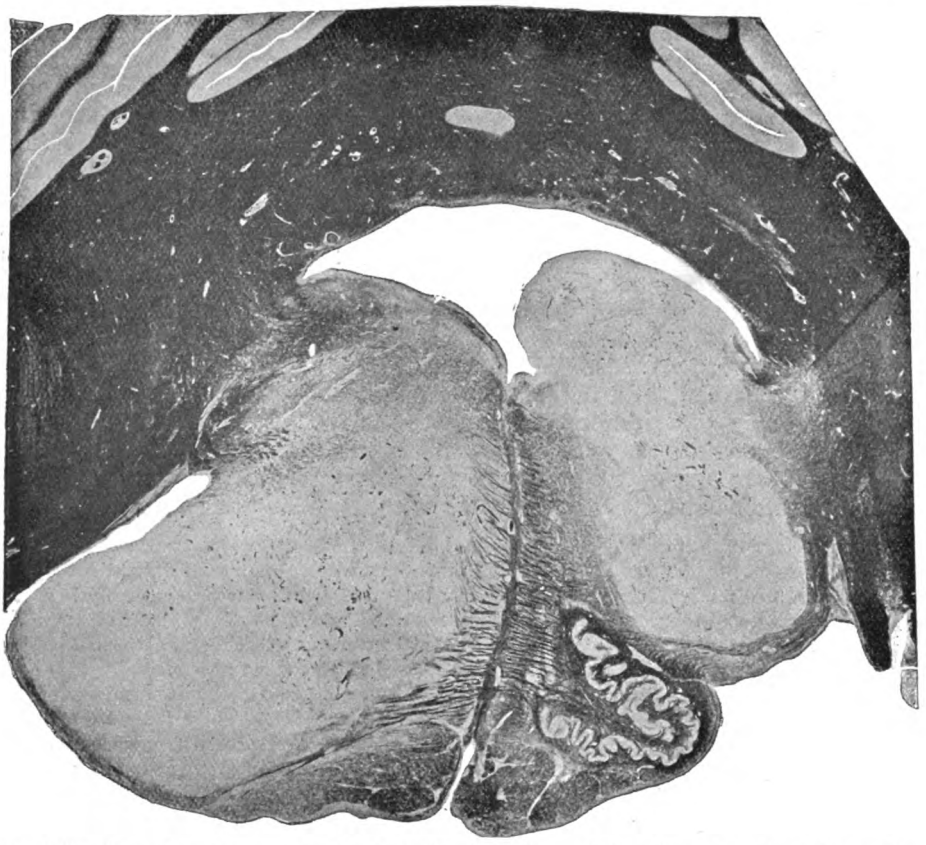

FIG. 339.—Two almost symmetrical foci of tubercle in the medulla oblongata. Case diagnosed by Oppenheim. (From photograph taken in the Neurobiological Institute.)

ventricular tumours. The earlier literature may be found in Audry (*Rev. de Méd.*, 1886) and the later in Henneberg (*loc. cit.*) and Hunziker (*Z. f. N.*, xxx.).

Tumours of the base of the brain are not as a rule difficult to recognise. If they arise from the bones, they may rupture through the nasal and pharyngeal cavities, causing hæmorrhage from these cavities, or they may be palpable at these points. They may also lead to the evacuation of hæmorrhagic masses, mixed with particles of tumour. If this does not occur, a local tenderness to pressure may sometimes be detected on the parts of the base of the brain which are accessible to palpation.

Choked disc and vomiting are very often absent. The diagnosis may otherwise be founded upon the signs of paralysis of the cranial nerves, as according to the site at which the tumour commences, various numbers or groups of these nerves may be compressed and proliferated, and may finally atrophy. I should like specially to state from my own experience that paralysis of the cranial nerves may for a considerable time be the only sign of tumour of the base of the brain, all the general symptoms of tumour being absent.

Tumours of the hypophysis [1] are specially characterised by disturbances of vision, *e.g.* bitemporal hemianopsia, unilateral or bilateral amblyopia or amaurosis, or unilateral amaurosis with hemianopsia of the other eye. The fundus of the eye remains for a long time normal. Atrophy of the optic nerves may develop later. Oculomotor paralysis is often present, and sometimes exophthalmos and anosmia. Diabetes mellitus or insipidus is not uncommon. The headache is often very slight. The relation between hypophyseal tumours and acromegaly is discussed in the corresponding chapter. For a description of the interesting conditions on the sella turcica which I have discovered by radiography, see below. In addition to acromegaly, these tumours seem to give rise to other disorders of the nutrition, especially to general adiposity (Fröhlich, Fuchs, Madelung, Erdheim), and a myxœdematous condition of the skin (Fröhlich, Fuchs, Erdheim, Thom). Loss of the hair, hypoplasia of the genitals (Babinski, Raymond, Cushing), a feminine appearance (Zak), dystrophia adiposo-genitalis (Bartels), and amennorhœa (Axenfeld,[2] Yamaguchi) have also been observed. A subnormal temperature is often mentioned.

In tumours of the median cranial fossa, the nerves which pass to the orbit are more or less completely atrophied. The most marked symptoms, however, are those due to affection of the trigeminus.

Lesion of the trigeminus may apparently cause exudation of fluid into the antrum of Highmore (a case of my own). In one of my cases of tumour of the median cranial fossa, the first objective symptom was protrusion of the eyeballs along with slight symptoms of paralysis in the oculo-motor region—the pupils being at the same time contracted. Headache had appeared eight years previously, and it was followed by sensory disturbance in the region of the first branch of the trigeminus, which was first manifested by absence of the corneal reflex, then by amaurosis of the corresponding, and temporal hemianopsia of the other eye, and finally by sensory and optic aphasia due to compression of the basal surface of the temporal lobe. The tumour was localised exactly. In another case it was remarkable that the pressure exerted by the tumour upon the first branch of the trigeminus near the bulb, produced contractions, apparently of a reflex nature, in the left facial nerve. A third case was specially interesting from the fact that whilst under our observation an oculomotor paralysis repeatedly developed and again rapidly disappeared.

Hartmann (*Journ. f. Psychol.*, vi.) has published a very important contribution to this question. He mentions the points which characterise the extra- and intra-dural site of the tumour, discusses its relations to extra-cranial tumours of the pharynx, etc., its localisation at certain favourite points in the median and posterior cranial fossa, the extension of malignant growths to both of these, etc. Benign, circumscribed fibromata of the median cranial fossa almost always arise from the trigeminus, those of the posterior cranial fossa from the auditory nerve. The former are com-

[1] For recent literature on hypophyseal tumours, see Fuchs, *W. kl. W.*, 1903, *Jahrb. f. P.*, xxvi.; Lannois-Roy, *Arch. gén. de Méd.*, 1903; Erdheim, *Sitzungsber. d. k. Ak. d. W.*, Wien, 1904, *Kl. M. f. Aug.*, 1905; Götzel-Erdheim, *Z. f. Heil.*, 1905; Erdheim, "Über Hypophysenganggeschwülste," etc., Wien, 1904; Madelung, *A. f. kl. Chir.*, Bd. lxxiii.; Hudovernig, *Z. f. N.*, xxxiii.; Berger, *Z. f. k. M.*, Bd. liv.; Yamaguchi, *Kl. M. f. Aug.*, Bd. xli.; Cuching, *Journ. Nerv. and Ment. Dis.*, 1906; Giordani, *These de Paris*, 1906; Bartels, *Z. f. Aug.*, xvi.; Bregmann-Steinhaus, *V. A.*, Bd. clxxxviii.; Frankl-Hochwart, *Z. f. N.*, xxxiv.; Marburg, *W. m. W.*, 1907.

[2] *D. m. W.*, 1903. It also occurs, however, in tumours at other sites, as Müller notes; see his communications on this subject in the *N. C.*, 1905.

paratively often associated with an extra-cranial new growth in the region of the parotid, of the angle of the jaw, etc. In contrast to fibroma, the malignant forms of sarcoma and carcinoma show a diffuse, superficial distribution, and their symptoms accordingly point to involvement of many of the nerves of the median and posterior cranial fossa. A limitation to the nerves of one cranial fossa is an indication of a circumscribed, knotty tumour. Carcinoma extends to the optic nerve, and soon gives rise to choked disc or amaurosis; the former hardly ever occurs except in tumours of the auditory nerve.

Symptoms due to compression of the cranial nerves arising from the pons and oblongata are specially characteristic of tumours of the posterior cranial fossa, etc. The cranial nerves may be affected on one or both sides. Tumours such as neuroma, fibroma, sarcoma, originate with comparative frequency in the auditory nerve (see Figs. 332, 333, and 340, Pl. vii.), and according to the cases cited by Oppenheim,[1] Monakow, Sternberg, Brückner, Sharkey, Moos, Hartmann,[2] and especially by Henneberg and Koch[3] and by Fraenkel and Hunt,[4] these give rise to a group of symptoms so characteristic that a local diagnosis can usually be established. I was the first in the year 1889 to direct attention to this typical clinical condition, being led to do so by a case which I had diagnosed with almost photographical precision. Henneberg and Koch give to these tumours the name of "neurofibroma of the cerebello-pontine angle." They are sometimes merely one of the symptoms of general neurofibromatosis (*q. v.*), or of central neurofibromatosis (Mossé and Cavalié, Oppenheim, Funkenstein). Hartmann speaks of tumours of the recessus acustico-cerebellaris. These growths are sometimes symmetrically distributed (see Fig. 341). Next to the auditory nerve, the trigeminus and vagus are most commonly affected.

The symptomatology as a rule is as follows: The disease generally begins with unilateral symptoms arising from the cochlear or vestibular nerves—buzzing in the ears, nervous deafness, vertigo, disturbances of equilibrium, "cerebellar seizures" (Dana), or vestibular attacks (Ziehen). These are followed by trigeminal symptoms, the corneal reflex on the corresponding side first being abolished (Oppenheim, etc.). The facial and vagus nerves are very often involved. These symptoms are soon followed by others which are due to compression of the cerebellum, pons, and oblongata. The most common of these are cerebellar ataxia, nystagmus, paralysis of conjugate deviation (which Uhthoff wrongly calls rare), and bulbar symptoms. The general symptoms, which precede the local ones, may run parallel with them; they may, however, only develop later and be very slight, so that the patient may be in a remarkably good general condition, may come himself for consultation, etc., but on the other hand these symptoms, in particular the headache and the changes in the fundus of the eye (choked disc), may be very marked. Vomiting is often, though not always present. The headache is frequently felt more acutely in the corresponding occipital and frontal region, and there is often localised tenderness to tapping or pressure and sometimes also diminished resonance in the occipital region (Oppenheim). Homolateral motor ataxia, especially in the arm, adiadococinesis, intention

[1] *B. k. W.*, 1890. [2] *Z. f. Heilk.*, xxiii., and *Journ. f. Psychol.*, vi. [3] *A. f. P.*, xxxvi.

[4] *Med. Rec.*, 1903. More recent contributions are those by Ziehen, *Med. Klinik*, 1905; Alexander and Frankl-Hochwart (*Obersteiner*, xi.); Funkenstein (*Mitt. a. d. Grenzgeb.*, xiv.; Seiffer-Borchardt-Oppenheim (*loc. cit.*); Stewart and Holmes (*loc. cit.*); Hills and Spiller (*loc. cit.*); Oppenheim, "Beitr. zu der Diagnostik und Therapie der Geschwülste," etc., Berlin, 1907; Alexander, *Z. f. k. M.*, Bd. lxii.; Bruns, *N. C.*, 1907; see also the section on treatment.

tremor, etc., may indicate an involvement of the cerebellum and the inferior cerebellar peduncle.

Tumours of the auditory nerve are distinguished from intrapontine growths by the fact (1) that choked disc is hardly ever present, and (2) that the paralysis of the cranial nerves is more often bilateral. Tumours outside the pons may, however, as I have shown, cause a bilateral paralysis of the cranial nerves during its later course.

These tumours cannot be definitely distinguished from new growths of the corresponding cerebellar hemisphere, especially when the latter have a basal site, but here the cerebellar and general symptoms are on the whole more marked and of earlier development.

We possess a valuable guide to local diagnosis in the *tenderness of the skull to percussion*. There are of course some cases in which a light tap

FIG. 341.—Bilateral neurofibroma of the auditory nerve. (After Henneberg.)

is felt as painful at every part of the head. I have hardly ever found this symptom except where the intracranial pressure was considerable and the bones of the whole skull attenuated by osteoporosis. The phenomenon is by no means dependent upon the site of the tumour. In other cases percussion is not felt as painful at any part of the skull. There remain a comparatively small number of cases in which percussion gives rise to pain only at a circumscribed site. This points to a local connection between the tumour and this part of the bone. Tumours arising from the cortex, the meninges, or the periosteum therefore, specially tend to produce this symptom, but it cannot be regarded as an absolutely positive sign.

The temperature of the skin of the part of the head above the tumour may be somewhat raised.

Percussion sometimes produces a tympanitic note or a cracked-pot sound in the part of the skull near the tumour, a symptom to which attention has been drawn by Suckling, Macewen, and especially by Bruns. This sign is particularly marked if rupture of the sutures has taken place.

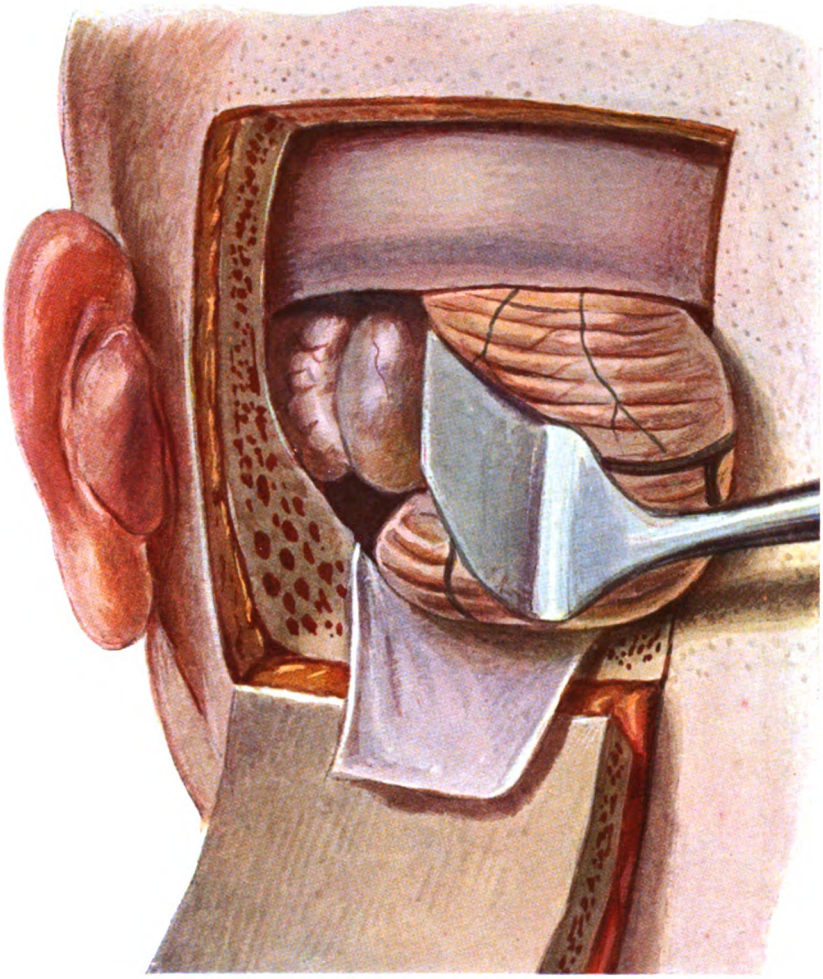

Fig. 340.

Tumour at the Left Cerebello-pontine Angle (Neurofibroma Acustici).

(From a sketch made during operation.) (Oppenheim and Krause's Case.)

Otto Schulze & Co., Edinburgh.

It may also occur under other conditions, and is constant in suckling infants. On the other hand I have occasionally found in tumours arising from the bones or the dura mater, that the percussion note was completely dulled and high-pitched over the tumour, in contrast to that at other parts of the skull. This has also been noted by Clarke and Landsdown. Williamson thinks he has found that in auscultation of the skull the whispering of the patient was heard more feebly above the tumour than at other parts. We should also mention here a communication by Phleps.[1]

With regard to the significance of the so-called cephalic bellows-sound, see the chapter on aneurism.

Indications for Diagnosis.—We must in the first place point out that brain tumours may remain latent until death. This is specially the case as regards tumours of slow growth or those which remain unaltered, such as psammomata, lipomata, cholesteatoma, and vesicular tumours.

Persistent and violent headache must always arouse suspicion of the existence of a brain tumour. But headache which is described as constant and persistent is very often due to other causes. The most important step, and one which should never be neglected, for the determination of the cause of the headache, is ophthalmoscopic examination. If choked disc is present, the diagnosis of brain tumour is very probably correct. We should then endeavour to exclude meningitis (serous, purulent, and tubercular), brain abscess, hydrocephalus, cerebral syphilis, and very specially nephritis, as these may lead to a form of retinitis which has a confusing resemblance to choked disc. If severe chlorosis is present, the existence of choked disc is not sufficient to refer an unyielding headache to brain tumour. A number of cases have been recorded in which brain tumour was simulated by chlorosis (Burton-Fanny, Jollye, Patrick, Engelhardt, Bannister, etc.). In chronic lead-poisioning, choked disc is not an infallible sign of cerebral tumour. Optic neuritis occurs also in multiple neuritis, both in its alcoholic and cachectic forms. It has been found in a case of carcinoma in which there was no tumour in the brain. The form of optic neuritis which accompanies multiple neuritis or is due to alcoholism, is usually the characteristic one of central colour scotoma. We should not forget that acute encephalomyelitis, myelitis, and disseminated sclerosis may be associated with neuritic symptoms in the fundus of the eye, and even with choked disc.

We should further remember that brain symptoms may occur in carcinoma without any pathological changes (Oppenheim, *Charité-Annalen*, xiii.), and that infiltration of the pia by cancer-cells may be the cause of the cerebral symptoms (Saenger). Interesting contributions to this subject have been made by Nonne, Warrington (*Journ. of Physiol.*, 1904), Weisenburg (*Univ. of Penns.*, 1905), and Finkelnburg (*Med. kl.*, 1906). The diffuse carcinomatosis and sarcomatosis of the meninges mentioned above should also be kept in mind.

The fact that there is a *pseudo-optic neuritis*—a congenital anomaly of development—should not be forgotten.

The diagnosis of brain tumour is by no means invalidated by the absence of choked disc or the fact that ophthalmoscopic examination reveals no abnormality. Choked disc may be absent at the beginning, or altogether when the growth is small, cystic, and situated on the surface

[1] *Arch. f. P.*, Bd. xliii. See also Koplik, *Med. Rec.*, 1906.

outside the cerebrum ; and it may not appear for a long time, if at all, when the tumour arises from the motor zone, the pons, or the oblongata. When this is so, our first endeavour should be to ascertain exactly the nature of the headache. We must seek for other causes, keeping in mind the toxic factors (arsenic, lead, copper, mercury, morphia, nicotine), arteriosclerosis, circulatory and digestive disorders—constipation is very often the cause of headache—and ascertaining specially whether hemicrania, hysteria, or neurasthenia are present. Although the headache of hemicrania is characterised by its periodicity, there are not a few cases in which it becomes to a certain extent permanent in the later stage of the illness. The diagnosis is to be established by careful inquiry into the history. The headache of hysteria, and still more that of neurasthenia, may give rise to difficulty in diagnosis. Knowledge of the presence of hysteria or neurasthenia determines many points, but not all. The case is most easily explained if we ascertain that the headache is chiefly dependent upon mental processes, that it is aggravated by any excitement, disappears when the attention is diverted, and that, although it is described as severe, the patient does not show the visible *impress of suffering*.

If the headache points to a tumour in the brain, whilst the results of ophthalmoscopic examination are negative, the diagnosis may be made from the evidence of the other indications. Stupor and drowsiness are exceedingly important signs, but they may be due to other causes, in particular to uræmia and other poisons, or to psychoses which begin with conditions of dreaminess and somnolence. Vomiting is a decisive symptom only when it is of a cerebral nature. Slowness of the pulse is of great importance, but the possibility of arteriosclerosis must be remembered, and also the fact that the pulse may be exceedingly slow at the height of an attack of migraine. This symptom also occurs in melancholia. Moreover, we must not forget that there are individuals who have congenitally a very slow pulse. Vertigo is a vague symptom. We must therefore inquire closely into its nature. The vertigo of brain tumour is independent of the imagination, of feelings of anxiety, etc. ; it comes on without any cause or reason, or it is constantly present. The various forms and causes of vertigo are thoroughly discussed in another part of this book.

Localising Symptoms.—These are of special importance as regards the diagnosis of brain tumour if they develop very gradually and progress steadily or in successive stages. This is not an axiom. A tumour may remain latent for a long time, or it may give rise only to general symptoms, until a hæmorrhage in it or a softening in its neighbourhood produce an acute, apoplectic onset of paralytic or irritative symptoms. This, however, is so rare that we may regard a gradual increase of the localising symptoms as the rule. Abscess must, of course, be excluded, as it may give rise to a similar course. The focal symptoms behave in the same way only in meningoencephalitis (syphilitic and tubercular), in the exceedingly rare cases of chronic brain softening (*q. v.*), and in localised senile atrophy of the brain (see p. 822). I[1] have described a number of cases in which the symptoms of a tumour of the Rolandic area in children or young persons disappeared spontaneously or after medical treatment (usually with iodide). The development was on the whole

[1] *B. k. W.*, 1901.

more rapid than it usually is in tumour. In diagnosing this disease, I was led to suspect a special form of encephalitis, but more especially a tuberculous meningoencephalitis, the *méningite en plaques* of the French writers (Chantemesse, Combe, Raymond, etc.). If the symptoms of a focal disease of the motor region appear in childhood, associated with the general symptoms of brain tumour, one should be very cautious in giving the diagnosis of a tumour in the strict sense of the word.

These observations have been greatly extended and supplemented by Nonne.[1] He has not merely observed complete disappearance of the tumour symptoms, usually under specific treatment, but he has been able also to show that in such cases there may be no apparent pathological basis. He has thus been able to establish the doctrine of "*pseudo-cerebral tumour.*" We are quite in the dark as to the nature of these conditions. Serous meningitis, which we were led to suspect, was absent in some cases, and no encephalitic foci could be discovered. We do not know whether we are dealing with a chronic toxic condition, with Reichardt's[2] brain-swelling, or with some process as yet quite unknown. Nonne's review of the subject undoubtedly contains cases of various kinds, to all of which the theory of pseudo-tumour need not be applied, but there remain other unobjectionable cases, which fit in well with Nonne's theory. The symptoms are usually those of a growth in the motor zone; there was a remarkable number of cortico-epileptic attacks, and sometimes a status hemiepilepticus (L. Müller); other cases simulated a neoplasm of the posterior cranial fossa.

Cases of this kind have since been communicated by Henneberg (*B. k. W.*, 1905, and *Charité-Annalen*, xxii.); Hoppe (*Journ. Nerv. and Ment. Dis.*, 1907); Saenger (*D. m. W.*, 1905); Vorkastner (*B. k. W.*, 1905); and Bonhöffer (*B. k. W.*, 1906). The cases published by Weber-Schultz (*M. f. P.*, xxiii.) are also worthy of note, as they again show how careful one should be with regard to this conception of pseudo-cerebral tumour. See also Knauer (*N. C.*, 1907) and Stertz (*N. C.*, 1907).

Reichardt's discovery that acute and chronic brain swelling, by which the weight of the brain, ordinarily about 14 per cent. less than the capacity of the skull, is considerably increased, may lead to choked disc and mental disorders, is a very remarkable one.

The symptom of cortical epilepsy must be very cautiously interpreted, as it also occurs in hysteria, intoxication (especially alcoholism and uræmia), paralytic dementia, etc.

Status epilepticus may also develop from intoxication, or it may be a symptom of genuine epilepsy or one of its varieties (L. Müller).

When the diagnosis of brain tumour has been definitely made, we must then seek to determine its *nature*, with the aid of the points given above, and to discover if there is tuberculosis in any of the other organs, or if there is any marked tendency to its development. It is not advisable to make use of tuberculin injections as a test for brain tubercle, especially as the results thus obtained (*e.g.* by Dupon and Ballet) are indefinite. This seems also to be the case as regards the more recent tuberculin reactions, but further experience is required on this point. We have to ascertain whether the patient has been infected with syphilis. This

[1] *Z. f. N.*, xxvii., xxxiii., xxxiv.; and *N. C.*, 1905.

[2] "Über die Entstehung des Hirndrucks bei Hirngeschwulst," *Die Heilkunde*, 1905, and *Z. f. N.*, xxviii.

possibility is by no means excluded by the absence of a definite history or positive physical signs. The clinical condition itself and the results of treatment may then furnish valuable evidence. Wassermann's reaction (sero-diagnosis) may be especially convincing. If there is cancer or sarcoma in other organs, there is every probability that the tumour in the brain is of the same nature. Lumbar puncture may assist in the recognition of syphilis, sarcoma, etc., but it is not advisable to use it for diagnostic purposes in cases of suspected tumour. One should never neglect to examine the lungs, as in many cases the somnolent patient fails to direct attention to these organs either by complaining or by severe coughing. It is also very advisable to extend the examination to the nasal and pharyngeal cavities, and to the antrum of Highmore. Teleangiectases or angiomatous nævi may, according to Kalischer, Lannois, Emanuel, Cassirer, and others, afford an indication of the angiomatous nature of the brain tumour. The existence of a general neurofibromatosis indicates the probability that the brain tumour is also of a neurofibromatous character. I have seen a number of cases of this kind. Uterine hæmorrhages and cachexia may direct attention to the deciduoma malignum (Marchand, Gottschalk, Siefert, etc.).

Further, it is necessary to determine exactly the *site of the tumour in the brain*. Although at an earlier date this local diagnosis was interesting merely from a scientific point of view, it has in our time acquired a practical importance which should not be underestimated. The advance in this respect has been enormous, increasing year by year, but even now it is impossible in the majority of cases to make a precise local diagnosis.

The earlier editions of this work and the monographs already referred to give the results of my own experience in this respect. It is, however, very difficult to work up all the material at my disposal, as I have lost sight of some of my patients or have only been able to examine them once, whilst in other cases in which the operation yielded negative evidence, no autopsy could be obtained, etc. There was also another, and not smaller number of cases, in which I could only give a provisional diagnosis, and finally, a large number in which surgical treatment was entirely out of the question on acount of the depth at which the tumour lay, or the entire absence of localising symptoms, or in which we could only recommend trephining for relief of the pressure (see below). Taking these facts into consideration, there remain for the purposes of definite diagnosis some ninety-five cases occurring in recent years, of which about eighty came under operation (attempted radical excision), the rest being examined post mortem. Errors as to general diagnosis were made only in four cases (three of hypdrocephalus, one of encephalomalacia). Mistakes of localisation were also very uncommon, an intramedullary or intrapontine tumour being twice found instead of the expected neurofibroma of the acoustic nerves, the tumour occupying in one case the left frontal instead of the temporal lobe, and in another case the right frontal lobe instead of the cerebellum. In the last case the patient had been admitted in the stage of complete blindness, and I had made the diagnosis with the greatest reserve, being only driven to operation by the deplorable condition of the patient. In another case I had at first thought that the tumour was probably situated in the left cerebellar region, but soon abandoned this view, assuming that, in addition to a tumour in the first temporal lobe or the central ganglia, there was a very extensive growth in the ventricle. I therefore advised decompressive trephining over the left temporal lobe and an exploratory puncture, which revealed tumour cells in its most anterior zone, near the frontal extremity of the hemisphere. The autopsy showed a large tumour of the left frontal lobe and chronic basal meningitis of the posterior cranial fossa, with involvement of the basal cranial nerves.

I may, therefore, say that the general and local diagnosis was correct in more than five-sixths of the cases at our disposal, and practically exact in nine-tenths. I have made it a rule, however, to give expression

in my diagnosis, which is usually a written one, to all the doubts and possibilities, and usually to indicate as merely probable, or very probable the site of the tumour in this or that section of the brain.

We should in the first place inquire as to the localising symptoms. The earlier these appear the more important they are. Even when a case has reached the somnolent stage before it comes under treatment, the focal symptoms may still help to establish the local diagnosis if it can be ascertained that they have been present from the very beginning. The tumour can be localised with the greatest certainty if the focal symptoms arising from the motor zone are very marked. We possess no definite criterion which would enable us to distinguish between cortical new growths of the motor zone and those of the subcortical centrum ovale. Tumours of the base, and especially those of the cerebello-pontine angle, the pons and cerebellum, can as a rule be localised with more or less certainty. The symptoms of frontal are sometimes so allied to those of cerebellar tumours, however, that errors are not always avoidable. Bramwell rightly places tumours of the right temporal lobe at the end of this series.

Even when the examination is made with the greatest care and experience, mistakes may be caused by various factors, viz., distant effects, injury of the tissue caused by hydrocephalus, œdema of the brain, the chronic meningitis which is sometimes present, swelling of the brain or toxic processes. These are associated with displacement of the brain which may often extend to distant parts of it, so that in tumours of the cerebrum, for example, the cerebellum and the medulla oblongata may be pushed into the foramen magnum. The dragging and displacement may involve the basal cranial nerves, especially the abducens of the same and even of the opposite side. Trigeminal symptoms are very rarely produced in this way, as Collier has shown (*Br.*, 1904).

It should be noted that a tumour situated near the middle line, *e.g.* arising from the central ganglia, may grow so far towards the other side and exert so severe a compression upon it that collateral focal symptoms may be the most prominent feature. The same effect may be caused by a complication with hydrocephalus, as in the cases of Dinkler, Ziegenweidt and Spiller. Cramer (*M. f. P.*, xviii.) and Weber (*A .f. P.*, Bd. xli.) have published some remarkable facts as to the symptoms of unilateral hydrocephalus.

Further, the skull should be carefully percussed, in order to discover if there is any part particularly sensitive to slight tapping.

Amongst the *external* signs of tumour on the skull, local prominence of the bone, which in rare cases is bulged outwards by the tumour, perforation, rupture of the sutures, which is not uncommon in youth, etc., may be of diagnostic value. Moreover, growths from the base of the brain may push towards the ethmoid bone, sphenoid bone, and the naso-pharyngeal cavity, where they may be palpated. It should also be remembered that *nasal hydrorrhœa*, *i.e.* escape of cerebro-spinal fluid through the nose, occurs in rare cases of brain tumour (Nothnagel, Wollenberg, Caskey, Freudenthal, Mignon, Oppenheim, etc.), although it has been noted chiefly in hydrocephalus (Magendie, Willis, Hugenin, Paget, etc.), and may possibly occur in healthy persons. In one case of tumour of the anterior cranial fossa, I have found the temporal artery of the same side enormously dilated and twisted, a symptom which disappeared after removal of the tumour.

Chloroma of the skull is, according to Pfeiffer (*M. m. W.*, 1906), characterised by the following symptoms; (1) painful exophthalmos with subsequent atrophy of the optic nerve, (2) disease of the ear and swelling of the temporal region, (3) anæmia or lymphatic leucæmia, with swelling of the glands and cutaneous hæmorrhages, (4) rapid course of the disease and youth of the patient.

Two diseases remain to be mentioned in regard to the differential diagnosis, viz., paralytic dementia and disseminated sclerosis.

A confusion between tumour and paralytic dementia is only possible in a few cases, which begin with persistent headache and attacks of irritation and paralysis, which do not at once reveal their "paralytic" character. If, however, we remember that choked disc does not occur in paralytic dementia, and further that the mental condition is characterised not by simple stupor, but by true dementia, that the attacks, even when they assume the appearance of cortical epilepsy, do not leave behind them any permanent paralysis developing in the course of the attacks, etc., we can hardly fall into this error. These symptoms are moreover followed by the speech disorder which is so pathognomic of paralytics, and others which cannot be discussed here. The chapter on paralytic dementia should be consulted as to the cyto-diagnosis, sero-diagnosis, and the presence of albumen in the cerebro-spinal fluid.

Cerebral tumour more frequently produces a condition allied to that of disseminated sclerosis (see p. 342). Confusion is most likely to arise when there is slight optic neuritis or when the choked disc has already passed into atrophy. Should nystagmus supervene, as it is specially apt to do in tumours of the cerebellum and corpora quadrigemina associated with tremor of a certain kind, as has been observed both in brain tumour and hydrocephalus, and should there be headache, vertigo, and unsteadiness in walking, one might naturally suspect the case to be one of sclerosis. I believe, however, that careful examination will always prevent this mistake. As a rule the visual disturbance is more severe in the atrophy arising from choked disc than in sclerosis. The tremor may indeed resemble that of sclerosis, but it is usually inconstant and much less definite, consists of fine, rapid oscillations, and is not so closely associated with voluntary movements. It most nearly resembles intention tremor in tumours of the cerebellum, of the cerebello-pontine angle, but it is then usually associated with ataxia. In a case under my care, in which the movements were typically those of the sclerotic type, there was also tremor in other groups of muscles during rest. Hebetude is not one of the symptoms of sclerosis, but it also may be absent for a long time in such cases of tumour, especially in those of the cerebellum. Convulsions, slow pulse, vomiting (which I have seen in very rare cases of disseminated sclerosis as a transient symptom during the giddy attacks), aphasia, cortical epilepsy, etc. etc., are signs opposed to sclerosis. It is doubtful whether the symptoms last named ever appear among those of disseminated sclerosis; in any case I should say that they are extremely rare. Finally, spinal symptoms are very seldom absent in disseminated sclerosis. Isolated cases have been reported, however, by Bruns and Nonne, in which the clinical features of disseminated sclerosis corresponded in every detail with those of cerebellar tumour.

Diffuse sarcomatosis of the cerebro-spinal meninges may give rise to symptoms very closely allied to those of cerebro-spinal syphilis (Nonne, Redlich,[1] Barnes,[2] Grünbaum. When the course is acute, it may resemble

[1] *Jahrb. f. P.*, xxvi. [2] *Br.*, 1905.

meningitis (Rindfleisch,[1] Heyde-Curschmann[2]), but the fever is of course absent. Further experience alone will show whether lumbar puncture on the one hand, by showing the presence of sarcoma cells, or lymphocytosis on the other, can enable us to arrive at a certain diagnosis (Philippe-Cestan, Oberthür, Rindfleisch, Dufour[3]). Diffuse meningeal sarcomatosis may also give rise to symptoms very unlike those of tumour and resembling those of syphilitic meningitis or paralytic dementia (Siefert).

Tumours of the cerebellum and fourth ventricle may produce symptoms resembling those of tabes dorsalis (Giannuli, Raymond). Mistakes in diagnosis will almost always be prevented by the presence of the general symptoms of brain tumour and the signs of increase in the cerebral pressure.

Acute encephalitis, which sometimes commences with optic neuritis, is distinguished from tumour by its acute, usually febrile development, and its further course. There are isolated cases in which no definite diagnosis can at first be made. On the other hand I have given a probable diagnosis of pontine tumour, in a case which was subsequently shown by the regressive course to be one of pontine encephalitis. In another case of this kind, improvement was afterwards followed by the development of symptoms of disseminated sclerosis. In yet another, shown by its subsequent course to be one of disseminated myelo-encephalitis, I had given the probable diagnosis of diffuse sarcomatosis of the cerebro-spinal meninges.

As regards chronic, progressive encephalomalacia, see p. 824. Senile atrophy of the brain may give rise to focal symptoms of slow development, but not to symptoms of brain pressure.

The chapters on hydrocephalus and abscess should be consulted as regards their diagnosis from brain tumour. We cannot too strongly emphasise the fact that serous meningitis may produce symptoms absolutely identical with those of brain tumour.

Tumours of various parts of the brain have in rare cases given rise to symptoms resembling those of myxœdema (Auerbach).

F. Krause (*B. k. W.*, 1903; *Beitr. z. kl. Ch.*, Bd. xxxvii.) has described a very rare case in which a developmental malformation of the skull in the region of the posterior cranial fossa, and a marked backward projection of the clivus Blumenbachii (*i.e.* basisphenoid and basioccipital) had, by reason of a great reduction in the space, simulated the symptoms of brain tumour. Rachitic hyperostosis of the bones of the skull may give rise to confusion with cerebral tumour (Homén).

Simple cysts of traumatic or unknown origin may produce a condition very like that of brain tumour, although the general increase of brain pressure is seldom so marked. They specially tend to cause focal symptoms resembling those of tumour when they are situated in the motor zone and its vicinity and in the cerebellum (cases of Hitzig, Cabot,

[1] *Z. f. N.*, xxvi.

[2] *Arb. aus path. Inst. Tübingen*, v. As regards the diagnosis these authors come to the following conclusions: If a cerebral disease, running a very chronic course, accompanied by paralysis of the cranial nerves, of slow development and with intermissions at the beginning, and subsequent variations in its intensity, corresponds in its general condition exactly with that of a brain tumour —although the severe symptoms of choked disc are often absent—and shows symptoms of a general chronic cerebro-spinal meningitis in the later stages, we are probably dealing with a generalised carcinomatous or sarcomatous infiltration of the brain meninges, even although a primary tumour is not discoverable during life.

[3] *R. n.*, 1904.

Taylor, Ballance, Auerbach-Grossmann, Lichtheim, Scholz, Oppenheim, etc.).

Lumbar puncture (see p. 761) may be of great value in diagnosing brain tumour from other diseases which are not associated with increase of cerebro-spinal fluid. If the fluid is increased and the intracranial pressure abnormally great, microscopical, chemical, and bacteriological examination may reveal the existence of purulent or tuberculous meningitis. If the fluid is clear and free from leucocytes and bacteria, these conditions are not indeed absolutely excluded, but the essential point is usually then to determine whether the case is one of hydrocephalus or tumour. This cannot be definitely ascertained from the condition of the fluid, but the amount of albumen which it contains is usually much greater in tumour than in chronic primary hydrocephalus or in serous meningitis, and according to Krönig, the number of lymphocytes is greatly increased in the latter. The increase in the number of lymphocytes may, according to the cases quoted on p. 766, decide the diagnosis as between cerebral syphilis and brain tumour. In sarcoma and carcinoma of the meninges corresponding tumour cells may be found in the fluid. Increase in the amount of albumen and a yellow colouring of the fluid may also be of diagnostic importance (Rindfleisch, Dufour, Schönborn, Grund [1]).

It should be noted, further, that lumbar puncture may yield negative results in brain tumour, especially when the cranial cavity and spinal canal are shut off from each other, or the cerebral ventricles are closed against the subarachnoid space. Under these conditions the brain troubles are apt to become more marked after lumbar puncture.

As regards *lumbar puncture* itself and its diagnostic importance, the reader should consult the section upon treatment in this chapter.

My attempts [2] to make use of *radiography* for the purpose of diagnosis have on the whole been a failure. I have indeed succeeded in showing experimentally that a tumour introduced into the cranial cavity and laid against the brain could be distinctly seen, but radiography applied to the living subject has yielded little positive result. Other writers (Church, Durante, Obici-Bollici, Londe, Mills and Pfahler,[3] Fittig,[4] Grunmach, Ballance-Zieman, Albers-Schönberg, Benedikt, Chudowsky) have been more fortunate, as in some cases at least they have been able to diagnose the site of the tumours from a shadow in the illumination. Although this is quite an exceptional occurrence, possible only under particularly favourable conditions, it is a fact that bullets have been thus located in the brain in a great number of cases (Schjerning, Eulenburg, Bergmann, Henschen, Chipault, Braatz, Holzknecht-Dömeny,[5] etc.). Moreover, in one of my cases in which the symptoms pointed to a *tumour of the hypophysis*, the skiagram revealed the interesting fact that the *sella turcica was unusually deep and dilated* (compare Fig. 342, *a* and *b* on Plate viii.).

I have reported this case at the meeting of the Gesellschaft für Psychiatrie und Nervenkrankheiten of 13th November 1899 (*Arch. f. Psychiat.*, Bd., xxxiv. S. 303), and showed the skia-

[1] *Z. f. N.*, xxxi. [2] *N. C.*, 1897; *B. k. W.*, 1897. [3] *Phila. Med. Journ.*, 1902.
[4] *D. m. W.*, 1903; "Fortschr. a. d. Geb. d. Röntgen.," vi.
[5] "Über Projektilextraktion aus dem Gehirn während Röntgendurchleuchtung," Vienna and Leipzig, 1905.

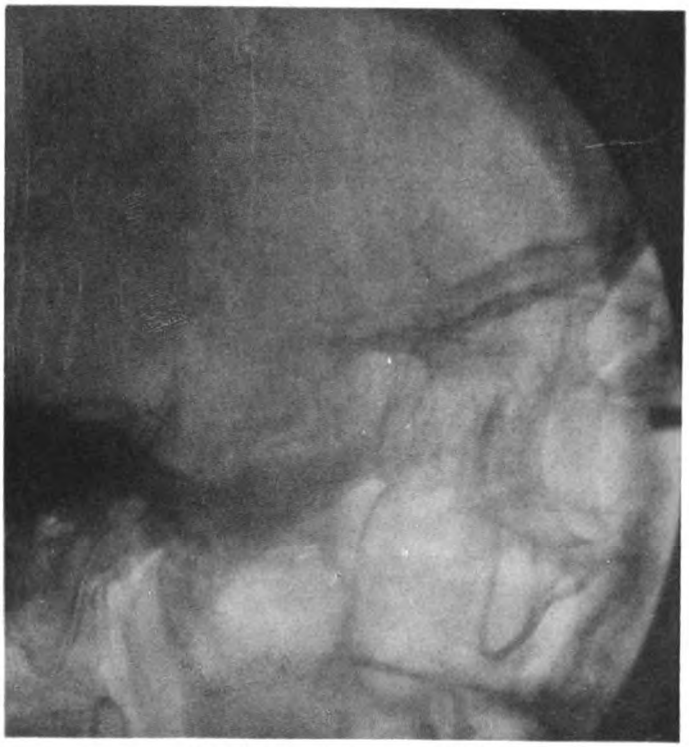

Fig. 342 A (compare with B).
Absorption and Enlargement of Sella Turcica in Tumour of Hypophysis (Acromegaly). Skiagram. (Author's case).

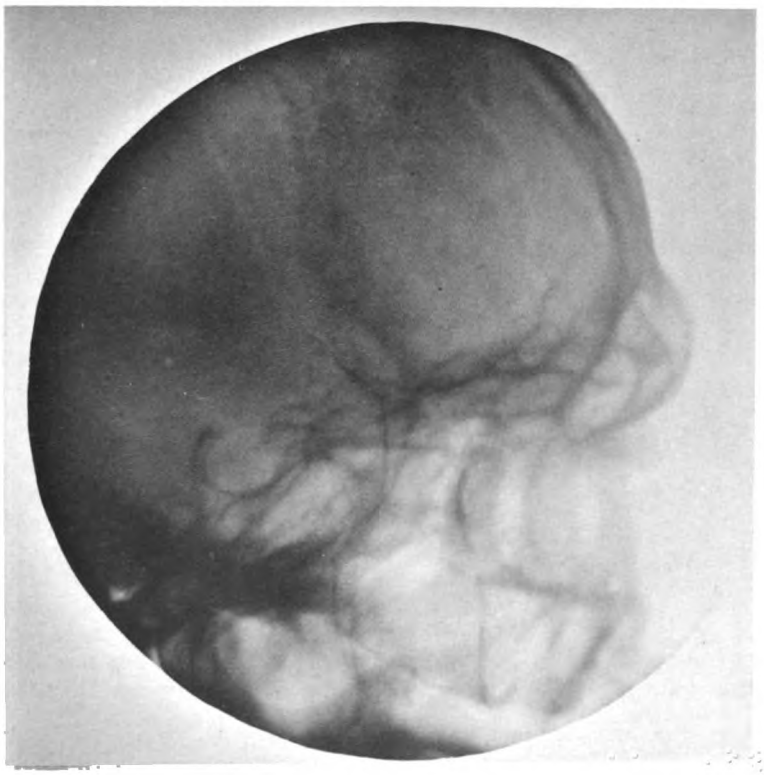

Fig. 342 B
Skiagram of Normal Skull. (Sella turcica).

Otto Schulze & Co., Edinburgh

gram in question, and I have also referred to it in the previous editions of this work. This communication has, however, I regret to say, been persistently ignored in contemporary French literature (Lannois et Roy, *R. n.*, 1903, etc.), and the case has been attributed to Marie.

My experience has been confirmed by these writers and also by Fuchs (*W. kl. R.*, 1905, *Jahrb. f. P.*, xxvi.), Erdheim (*loc. cit.*), Albers-Schönberg (*D. m. W.*, 1904), Kraus (*B. k. W.*, 1905), and many others. See also Schüller-Robinsohn (*W. kl. R.*, 1904), Schüller (*Fortschr. auf Geb. d. Röntgen*, ii., Ergänzungsband, and *W. m. W.*, 1908), and in the monograph by Fürnrohr. Erdheim has discussed the variety of changes in skiagrams of the sulla turcica and their relation to the special site of the tumour. It should not be forgotten, however, that hydrocephalus (or tumour at a distant site) may also lead to bulging and erosion of the sella turcica, as I have myself seen, and that this condition must therefore be subjected to very careful and critical consideration.

Pfahler is of opinion that radiography is most likely to give a positive result in cases of fibroma, osteofibroma, and ossified tubercle.

Etiology.—The causes of brain tumour are practically unknown. It is certain, however, that a wound of the skull may form the basis of a tumour. The first symptoms often appear directly after the injury, but more commonly after a longer or shorter interval.

A case reported by Leszynsky (*Journ. Amer. Med. Assoc.*, 1907) is of special interest in this respect, as a piece of bone from the inner table of the skull was found in the post-traumatic tumour. It has been found impossible to prove the view that only certain kinds of new growths, and those located at certain sites, may have a traumatic origin (Adler). Bünger thinks, however, that osteoma and sarcoma are specially due to this cause. The traumatic etiology of endocranial growths has been explained by various theories (Hansemann, Ribbert, etc.), which are mainly based on Cohnheim's teaching.

Some kinds of tumour have their origin in congenital defects of development. These include angioma, cholesteatoma, neurofibroma, certain kinds of cysts, etc., and possibly also glioma. Cicatrices may probably give rise to the formation of tumours.

The *infective* nature of some forms of tumour, and the *metastatic* character of others indicate their etiology.

Course and Prognosis.—The course of a brain tumour is almost always a chronic one. The disease as a rule covers a period of 2 to 4 years, but there is nothing extraordinary in a duration of 5 to 6 and even 10 years. I have observed this prolonged course most frequently in tumours of the cerebellum, the right frontal lobe, and the base of the brain. Experience also shows that the disease is not usually so soon fatal in young persons, in whom the walls of the skull are still capable of yielding to the pressure, as in adults. The course is as a rule more protracted in cases of solitary tubercle, glioma, or osteosarcoma, than in soft sarcoma and especially in carcinoma. It has been unusually prolonged in a few cases of osteoma, cholesteatoma, and angioma cavernosum. Cases have even been described in which such tumours have been in existence for twenty, thirty, and forty years (Joffroy-Gombault, Edinger, Mingazzini).

It is only in rare cases that tumours remain latent for a long time and then give rise to acute and even fulminating symptoms which are rapidly fatal. I have seen one case, in which a young healthy woman had complained for nine years of periodic headache. This was regarded as hemicrania, until it increased in severity and persistence, and the symptoms of a brain tumour (aneurism ?) suddenly appeared within two or three days and proved rapidly fatal. In several of my cases of pontine tumour, the disease showed an acute or subacute onset; in a few of these

it was so apparently a direct consequence of an injury that the assumption of a vascular process was the only possible one. Bruns, Hudovernig, Laslett, and others report similar cases. Prolonged intermissions sometimes occur.

Sudden death has been most often observed in tumour of the posterior cranial fossa.

The *prognosis* is comparatively favourable in *syphilitic* brain tumours, but otherwise it is exceedingly grave. *Spontaneous recovery* is possible in echinococcus, cysticercus, and in aneurism. Some writers are of opinion that tubercle is also capable of spontaneous cure by retrogressive metamorphosis. A number of cases (Wernicke, Gowers, Knapp, Baginsky, Sternberg, Kallmeyer, Williamson, Halban-Infeld, Foa,[1] Naegeli [2]) have been explained in this way, but it is exceedingly difficult to prove this view. The fact that these tumours may become calcified and ossified, as Simon and others have shown, by no means excludes the possibility of spontaneous recovery.

A case reported by Kirnberger [3] also points to this process. See also Zappert (*Obersteiner*, 1907). It is doubtful whether the possibility of spontaneous recovery from angioma (Swoboda, *W. kl. R.*, 1905, etc.) also applies to angioma of the brain.

I have myself thought that there is reason to believe on clinical grounds that localised tubercular meningoencephalitis may be capable of spontaneous cure by cicatrisation. Williamson and Roberts report a case in which a myxomatous tumour remained stationary for forty-six years.

In exceptional cases periodic improvement is caused by some part of the contents of the ventricle being evacuated, usually through the nose, after erosion of the skull.

Treatment.—Should syphilis be present, or should the symptoms point to the tumour being of a syphilitic nature, the method of treatment is clearly indicated. We cannot, however, state too emphatically that even a slight suspicion of its presence not only justifies, but urgently demands *anti-syphilitic measures*, and that these should be energetically carried out (large doses of iodide being given). If, however, the clinical condition shows no indication of brain syphilis, and if the history and physical examination of the patient in no way warrant this suspicion, time should not be unduly lost in anti-syphilitic treatment, if the site of the tumour is such that a well-timed operation might be expected to lead to recovery. This, it is true, is seldom the case. On the other hand systematic administration of large doses of iodide of potassium should never be omitted, as considerable improvement and intermissions have been observed to follow the use of this drug in a few cases of non-syphilitic tumour (aneurism, glioma, tubercle, sarcoma, cyst) (Wernicke, Oppenheim, Clarke, Böttiger, etc.).

Surgical treatment of brain tumour represents one of the most remarkable advances in treatment. It is not very long since every patient suffering from brain tumour was inevitably left to die. Wernicke had, it is true, even in 1881, referred in his *Lehrbuch der Gehirnkrankheiten* to the indications for surgical treatment, and shortly after reported a case in which a tubercular abscess of the occipital lobe was found and evacuated after the skull was opened. Subsequently, however, it was shown,

[1] *R. n.*, 1903. [2] *Schweiz. Corresp.*, 1907. [3] *Inaug.-Dissert.*, Freiburg, 1898.

especially by English and American surgeons (Bennet, Godlee, Macewen, etc.), to be possible to remove a brain tumour without endangering the life of the patient, and Horsley in particular proved that complete recovery might be attained by surgical removal of new growths in the brain which could be localised at a sufficiently early stage. This celebrated neurologist and surgeon has since then remained the master of this subject, and he may well be proud of his great experience and success. I have also to report a number of excellent results from the cases which I have treated with Bergmann, Borchardt, and especially with Krause. Wider experience has shown, certainly, that a *very small* number of cases are accessible to surgical treatment, and that it also involves considerable danger to life, but there is no doubt that progress is being steadily made in the development of this subject.

According to Starr's statistics of 1171 cases, some 7·5 per cent. may be regarded as accessible to operation. In a later account (*Journ. Amer. Med. Assoc.*, 1904) he regards 10 per cent. of the cases as adapted to operation, the results being successful in 5 per cent. Ferrier, Oppenheim, Walton, and Paul (*Journ. Nerv. and Ment. Dis.*, 1905) have come to similar conclusions.

The indications for operation, according to Bergmann and others, are somewhat as follows : The tumour must be situated in the cerebral cortex or immediately below it, and must lie in the external convex surface of the hemisphere ; it must not extend diffusely through the tissue, but must be capable of enucleation. Even infiltrating tumours have, however, been successfully removed. There must be a probability that the tumour is an *isolated* one and not of malignant or metastatic character. Nor should it be too large. Finally, the symptoms must furnish an indication as to its site.

Practically we may state the case thus : It is chiefly tumours of the motor zone which can be localised and diagnosed at a sufficiently early stage. If we consider the cases upon which operation has been performed, and especially those which have been successful, we find that the great majority belonged to this region. Bergmann's statistics of 116 cases show that in eighty-seven instances (75 per cent.) in which the growth was found at the expected site and successfully removed the tumour occupied this region of the brain. According to Bergmann, brain surgery is thus surgery of the central convolutions. Subsequent experience has, however, widened the field of operation, as tumours have been successfully removed from various other regions of the brain, viz., from the frontal, parietal, temporal, and occipital lobes, and from the posterior cranial fossa, whilst on the other hand in regard to tumours of the motor zone diagnostic errors have become more common. I have already referred to this change in my Stuttgart review. But in any case the motor region (and the cerebello-pontine angle) is the most favourable field for exact localisation of the tumour. The sooner a diagnosis can be made, the smaller will the tumour probably be, and therefore the better will be the chances of its successful removal. A case reported by Erb shows, however, that even large tumours can be successfully excised, or that the operation may be several times repeated if the excision has been a partial one. Bramann has removed a tumour weighing 280 gr. from the frontal lobe with the best results, and Poirier had a similar case. In one case upon which I operated along with Köhler —the first case of brain tumour treated in this way in Germany—we

only succeeded in removing part of the tumour, and thus procured very considerable improvement lasting for six months, during which time the patient gave birth to a living child.

We now know that recovery from a successful operation may be a complete one, and our observations have extended in several cases over ten to twelve years and more. I have lately been able to present one of my patients, from whom a brain tumour had been removed eight years previously by Bergmann.

Repeated operations have in many cases been successfully performed (Cabot, Beevor, Ferrier, Ballance, Kosinski, Stieglitz - Gerster), for the most part for cysts or cystic tumours. Ballance has operated five times in one such case. Three years after a successful operation on a cerebellar glioma, Jaboulay[1] had to open a cystic hernial sac which had appeared, and to make a permanent fistula for the escape of the fluid. Mitchell, Clark, and Landesdown, having found that after removal of a sarcoma all the symptoms did not disappear, diagnosed a second tumour and removed it by operation. Krause and I have in one case excised a cyst from each cerebellar hemisphere with entire success. Neisser and Pollack report one case in which a meningeal cyst in the cerebellum was punctured six times within a year and a half, the severe symptoms recurring every time. In one case where I had diagnosed a tumour in the paracentral lobe, Bergmann found five tumours instead of one in this region, and removed them all at one operation.

As I have already stated, other focal symptoms not of a motor character may indicate the necessity for surgical treatment. Should the general symptoms of tumour be associated with signs of a pure and well-defined form of aphasia, and should it be ascertained that this symptom was present at the beginning of the disease, although to a slight extent, and that it has since then gradually developed, we are justified in opening the skull in the region of the third left frontal or the first temporal convolution, and in removing the tumour which may be at once evident, or may require to be looked for. Operation has already been performed at this site in sixty-six cases, which I have collected from the literature, and in eighteen of these with good results. In addition to these I can report several cases of my own, amongst them a recent one in which the result was very satisfactory. In one of these the symptoms included the mental disorder (facetiousness) described above, which pointed to the localisation of the tumour in the right frontal lobe or its neighbourhood. It was found at the expected spot and its removal was followed by complete recovery, the mental disorder entirely disappearing. Bayerthal, Devic-Courmont, Friedrich,[2] and others report recovery of the mental symptoms after excision of a brain tumour.

Successful operations on the *parietal* lobe have of late years been reported in a considerable number of cases, especially by Keen, Mills and Spiller, Oppenheim-Borchardt,[3] and it has become more and more possible to make the local diagnosis in this region (compare p. 628).

Operations with good results have also multiplied in the *occipital* lobe (Thiem-Cramer, M'Kennan, Frazier, Oppenheim-Krause,[4] Lichtheim, Kümmel,[5] Bruns,[6] etc.). The guiding symptom has usually been

[1] *Lyon Méd.*, 1905.
[2] *Z. f. Chir.*, Bd. lxvii. See also a case by Armour (*Br.*, 1907).
[3] *B. k. W.*, 1906, Nr. 30.
[4] *B. k. W.*, 1906.
[5] *N. C.*, 1907.
[6] *D. m. W.*, 1907.

hemianopsia. The tumour in such cases has mostly been a cyst, but in one particularly successful case of Krause's and mine the tumour was a solid one.

Sommer, Heidenheim-Edinger, Knapp,[1] Pfeifer,[2] and Oppenheim[3] report operations on the *temporal lobe.*

By far the greatest change of view is that regarding the indications for operation in tumours of the *posterior cranial fossa.* Although cases of cure and alleviation had been reported from earlier times by Macewen, Horsley, Parkin, Beevor-Ballance, Schede, Terrier, Murri, Saenger, Collins-Brewer, and others, the percentage of deaths following immediately upon the operation was so large that Bergmann and I would have excluded cerebellar tumour from surgical treatment. In the meantime, however, the diagnosis of neurofibroma of the auditory nerve has become more and more developed, its usually benign character has been more fully recognised, and Monakow has included it among the tumours in which operation was indicated. Hartmann, Hitzig, Oppenheim, Seiffer, and others maintained an opposite opinion. However, the experience gained during these last few years has made it evident that growths of this region and of the cerebellum itself must be regarded as accessible to surgical operation. I would refer to communications of this kind by Horsley,[4] Schultze-Schede, Ballance, Oppenheim-Borchardt,[5] Oppenheim-Krause, Stewart-Holmes, Fraenkel-Hunt,[6] Putnam-Waterman,[7] Lichtheim,[8] Scholz,[9] Winkler-Rotgans, Neisser, Auerbach-Grossmann,[10] Anschütz (*D. m. W.*, 1907), Förster (*B. k. W.*, 1908), and many others. Out of twenty-five cases of operation in this region under my own observation, recovery took place in three, considerable improvement in three, and death in the remaining cases.

The prospects are specially favourable in cases of cerebellar cyst; according to Borchardt there were thirteen cures out of fourteen operations. That the results of operation on the cerebellum are lasting is shown, *e.g.* by a patient upon whom Horsley operated eleven years ago. It must be stated, however, that even now failure is still much the most frequent result, as we see from the experience of Starr, Sachs,[11] and others.

Retrobulbar tumours or those growing into the orbit have often been successfully excised.

Tumours of the base of the skull in the region of the middle cranial fossa, *e.g.* those arising from the Gasserian ganglion, may be surgically treated. I have seen Krause perform this operation in one of our cases, the result being a remission of many months' duration. We may refer also to similar communications by Partsch (*D. m. W.*, 1904), Hoffmeister-Meyer (*Z. f. N.*, xxx.), Friedrich (*D. m. W.*, 1906), and Eiselsberg.

An attempt has several times been made within recent years to operate upon hypophyseal tumours, sometimes from the naso-pharyngeal cavity, sometimes by means of Krause's operation, etc. I can only here refer to the cases of this kind reported by Braun,[12] Krause, Horsley,

[1] "Die Geschwülste des rechten und linken Schläfenlappens," Wiesbaden, 1905.
[2] *A. f. P.*, Bd. xlii.
[3] *Loc. cit.*
[4] *Br.*, 1903, and *Brit. Med. Journ.*, 1906.
[5] *B. k. W.*, 1907; *A. f. kl. Chir.*, Bd. lxxxi.
[6] "Contributions to Surgery of the VIII.," etc.
[7] *Journ. Nerv. and Ment. Dis.*, 1906.
[8] *D. m. W.*, 1905.
[9] *Mitt. aus d. Grenzgeb.*, xvi.
[10] *Mitt. aus d. Grenzgeb.*, xviii.
[11] *Med. Rec.*, 1906; *Beitr. z. kl. Chir.*, Bd. l.
[12] *Z. f. Chir.*, Bd. lxxxviii.

Schloffer,[1] Loewe,[2] Eiselsberg-Frankl-Hochwart,[3] and Hochenegg,[4] and say that, so far, our experience goes to show that the results are but incomplete and transient. Further attempts, however, appear to be justifiable. Hochenegg has seen the symptoms of acromegaly disappear after operation.

Horsley has operated ten times upon the hypophysis, and has introduced the laryngoscope into the sella turcica. Just as I write, Krause has excised a tumour of this region in a case under the care of myself and Schoeler, but the result has proved fatal.

Finally, Ballance has described a remarkable case in which he succeeded in removing a solid tumour from the optic thalamus with good results.

The diagnosis and treatment of brain tumours has been not a little advanced of late years by the adoption of *Neisser's brain puncture.* With regard to the technique, results, and value of this method, we can only refer to the most essential points, but the details may be found in the original papers of Neisser-Pollack,[5] Pfeifer,[6] Pollack,[7] Weintraud [8] and Ascoli, and from the corresponding reports of the first Congress of the Society of German Neurologists.[9]

Neisser, under local anæsthesia, directly perforated the skin, soft parts, and bones with the electric borer. It is best to use a graduated platinum-iridium needle, 7 cm. in length and 1·06 mm. thick (1·3 according to Pfeifer), with an oblique point. The needle, provided with a stilette, is inserted for a depth of 3 to 4 cm. The stilette is withdrawn, the syringe (Luer's syringe with an inserted glass top) is applied. After aspiration its contents are squirted into a vessel which is at hand. This manipulation is repeated, the needle being gradually withdrawn.

In this way one can directly obtain the contents of the cyst, or portions of tissue, which can be examined either fresh or after being hardened. The latter method, specially improved by Pfeifer, has been of much service. We may conclude from the descriptions of Neisser and others that puncture may save the patient's life by drawing off the fluid and may directly lead to cure of the cyst.

Neisser and Pollack have given exact directions as to the site to be selected for the puncture (see Fig. 326, p. 874). For the frontal region, they recommend two points on a line drawn backwards through the middle of the superior margin of the orbit, parallel to the middle line, one 4 cm., the other 8 cm. above the supraorbital margin; for the cerebellum the middle of the connecting line between the external occipital protuberance and the tip of the mastoid process (below the transverse sinus). As regards the points on the temporal lobe, see p. 873. The inferior cornu of the lateral ventricle will be found here, 1 cm. above the insertion of the ear, at a depth of about 4 cm. (Pfeifer). For puncture of the central lobe, it is recommended that the præcentral sulcus and the anterior central convolution should be determined by means of Kocher's cyrtometer. The inferior central region is least well adapted for puncture on account of the vessels. Kocher indicates a point 2·5 to 3 cm. below the bregma as the most favourable site for puncture of the lateral ventricle. When this cannot be definitely felt, it can be quickly found by drawing a line from a point immediately below the nose to the orifice of the auditory meatus and erecting a line perpendicular through the latter; the bregma lies at the point where this divides the sagittal line.

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[1] *B. k. W.*, 1905, 1906, and *W. kl. W.* 1907. [2] *B. k. M.*, 1907.
[3] *W. m. W.*, 1907, and "Verhandl.-Ber. d. Ges. deutscher Nervenärzte," Dresden; *Z. f. N.*, xxxiv.
[4] *D. m. W.*, 1908. See also discussion in *B. k. W.*, 1908.
[5] *Mitt. aus d. Grenzgeb.* xiii. [6] *A. f. P.*, Bd. xliii., and *Jahrb. f. P.*, xxviii.
[7] *Mitt. aus d. Grenzgeb.* xviii.
[8] "Therapie d. Geg.," 1905. See also Lichtheim, *D. m. W.*, 1905. [9] *Z. f. N.*, xxxiv.

We regard this method as justified and valuable in cases in which the local diagnosis of a tumour can only be made with a certain degree of probability, but in which there is still doubt as to its exact site, *e.g.* in cases where it cannot be determined whether the tumour lies in the occipital or temporal lobe, in the cerebellum or the frontal lobe, or whether it is situated more anteriorly or posteriorly within the occipito-parietal region, etc. Puncture may also be used when it is important to ascertain the nature of the tumour before performing the radical operation. Finally, one is justified in puncturing, at least when the local diagnosis is certain, in order to establish the general diagnosis, to determine whether for instance we are dealing with a tumour, a softening, or a hæmorrhagic pachymeningitis.

We would strongly deprecate any random puncturing here and there in the different sections of the brain, and we consider that Pfeifer especially, in spite of his service in the matter, has gone too far. Although we ourselves have found that the method is as a rule an easy one and is well borne by the patients, with whom one can converse during the operation, yet it is not free from danger (hæmorrhage, etc.), as shown indeed by Neisser and Pollack's cases. I have also become convinced that a brain which has undergone puncture at different points becomes an impaired organ with a diminished power of resistance.

Puncture should not determine a diagnosis ; it should merely confirm it when a blank point has been reached.

The more severe symptoms, especially the general ones, may entirely disappear shortly after surgical removal of a tumour. The effect of excision of the tumour (and even of simple opening of the skull) is specially apparent upon the choked disc, as it often disappears within the first few days after the operation. In one of our cases this occurred within twenty-four hours, and in a patient of Devic-Courmont within forty-eight hours after the operation. On the other hand I have once seen an amblyopia which was present, develop into amaurosis immediately after excision of the tumour, but this lasted for a short time only and was followed by steady improvement. The affection of the optic nerve is by no means always cured in this way, however. The monoplegia, hemiplegia, hemiataxia, hemianopsia, apraxia, etc., may disappear very soon after the operation, as I have often seen. It is obvious on the other hand that fresh symptoms, *e.g.* paralyses, aphasia, may supervene ; this has been observed in a good many cases, but has usually been a merely transient effect of the operation.

Although we must be specially careful in judging the statistics on these points, we may mention a few of these. Starr (*Journ. Nerv. and Ment. Dis.*, 1903) has collected 365 cases in which a brain tumour had been surgically treated, and he states that the tumour was not found in 111 cases, and was found but not removed in 27. Of the cases from which the tumour was removed 59 died, and 168 were cured (? refs.). The statistics of Pilcz apply to 94 cases, in which there was an operation. The tumour was found in 61 of these, *i.e.* in 64·9 per cent. ; recovery followed in 30 (31·8 per cent.) ; considerable improvement was obtained in 14 ; 17 died after the operation, and in 39 cases the tumour was not found. 21 of the latter cases ended fatally, whilst 12 were improved. Of the 44 cases which were cured or improved, 32 were tumours of the central convolutions. Ferrier regarded 13 per cent. of his cases as cured, whilst 37 per cent. died within a few days after the operation.

Bergmann's statistics, which I have extended, included 371 patients whose skulls had been opened under the diagnosis of brain tumour. Of these 140 (37 per cent.) died from the operation, *i.e.* during the latter or within a few days or weeks, etc. In 88 (23-24 per cent.) there was

recovery or great improvement. In 111 (28-30 per cent.) the operation had little or no result (these include some of the palliative and exploratory operations). Of the 166 cases of radical operation, 82, *i.e.* about 50 per cent., recovered.

Duret's statement, that 258 out of 400 cases of operation led to cure or great improvement, is certainly not in accordance with the facts. Knapp (Contrib. from the *Mass. Gen. Hosp.*, Boston, 1906) has published a very comprehensive statistical account, which compares the results at the various epochs of brain surgery. He shows that success has gradually increased and mortality diminished. In contrast to the very unfavourable reports of F. Schultze (*Mitt. aus. d. Grenzgeb.*, xviii.) we have Horsley's excellent results (*Brit. Med. Journ.*, 1906). It is true that he gives us no information as to the extent of the material at his disposal, the percentage

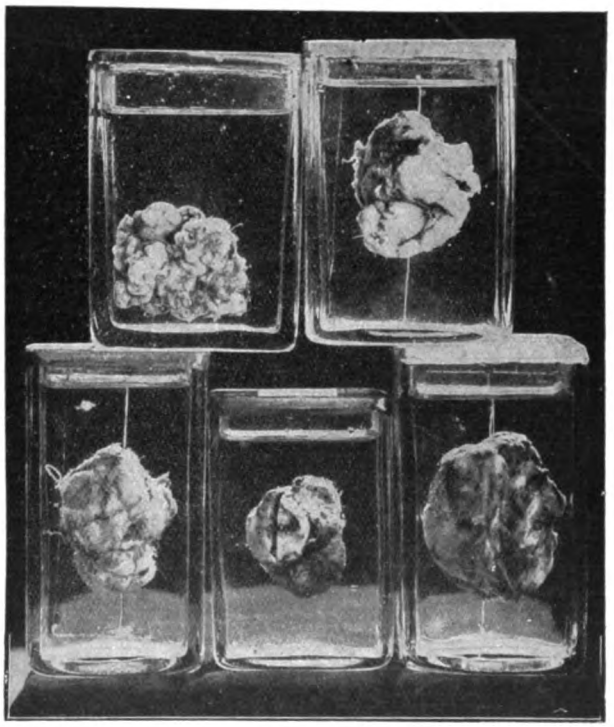

FIG. 343.—Tumours taken from the living brain in five different cases (reduced to a fourth). (Oppenheim.)

of errors in diagnosis, or failures in operation, but he leaves us in no doubt as to the great number of cases in which he has been successful.

Within the last few years I have had some seventy-five cases with recovery, which, however, was perfect and complete only in five of these, some of the paralytic symptoms persisting in all the others. Figs. 343 and 344 give some idea of the tumours removed in some of our cases (see also Figs. 329 and 340, Plates vi. and vii.).

The monographs already quoted and the text-books should be consulted as regards the technique of the operation. The article by Horsley in the *Brit. Med. Journ.*, 1906, in particular contains very valuable data.

Should the brain tumour be inoperable—and it is so in the majority of cases—the question then arises whether there is any method of symptomatic treatment which would alleviate the most distressing troubles,

the headache in particular. The drug which is of most use in this respect is *morphia*, and in grave and hopeless cases one may use it without any misgivings. I have occasionally seen marked diminution of the headache from the use of *antipyrin*, pyramidon, etc. The other narcotics may be tried alternately with the morphia. An ice-bag applied to the head sometimes soothes the pain. No objection can be made to local blood-letting, or the application of several leeches, if the pain is very severe. Hot foot-baths, aperients, etc., may also have a beneficial effect.

Surgery may, however, be of some use even in these incurable cases. It has been found that simple *trephining* of the skull with opening of the dura and withdrawal of the cerebro-spinal fluid may materially lessen the intracranial pressure (Weir, Horsley, Bramwell, Macewen, Gussenbaur, Bruns, Oppenheim, Taylor, Chipault, etc.). It is sufficient to alleviate the *headache*, *choked disc*, stupor, and the other pressure symptoms

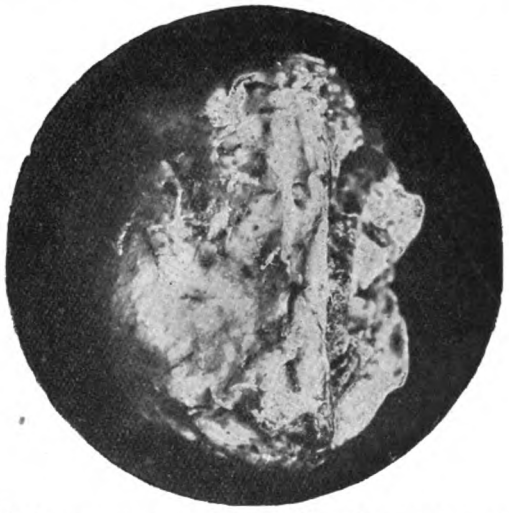

FIG. 344.—Brain tumour removed during life. Natural size. (Oppenheim.)

for a long time. I know many cases in which a result of this kind was obtained. In one of these, in which it was impossible to remove the whole tumour, which subsequently grew to a great size, the choked disc was absent until death. On the whole, however, I cannot speak of the benefits of decompressive trephining with the same enthusiasm as Horsley,[1] Spiller-Frazier,[2] Saenger,[3] Paton,[4] Fürstner,[5] Cushing,[6] and others have lately done, as in some of our cases there was no alleviation or improvement except diminution of the headache, and in some of these the result was rapidly fatal.

Horsley recommends that trephining should be performed at the supposed site of the tumour. This is certainly right in every case if there is any possibility of a radical operation, but, if not, I think it advisable to select the region of the right temporal lobe, as the prolapse which sometimes occurs would there affect a part of the brain which is of com-

[1] *Loc. cit.*
[2] *Loc. cit.*, and *Journ. Amer. Med. Assoc.*, 1906.
[3] *B. k. W.*, 1906, and *Kl. M. f.*, Aug. 1907.
[4] *Ophthal. Soc.*, 1905.
[5] *A. f. P.*, Bd. xli.
[6] *Loc. cit.*, and "Surgery, Gynæc.," etc., 1905.

paratively little physiological importance. Saenger has accepted this advice. Cushing would make use of the temporal muscle for covering the site of the operation, in order to check the bulging of the brain. It would certainly be a practical advantage if the prolapse could be averted. Frazier avoids making an incision in the dura, but it is doubtful whether the intracranial tension will otherwise be sufficiently diminished. Horsley is under the impression that mere opening and exposure will cause a glioma to disappear, but I on the contrary have sometimes thought that the growth of a diffuse glioma became more rapid after an attempt at excision.

I cannot agree with Horsley's opinion that the appearance of optic neuritis should be an unconditional and immediate indication for tre-

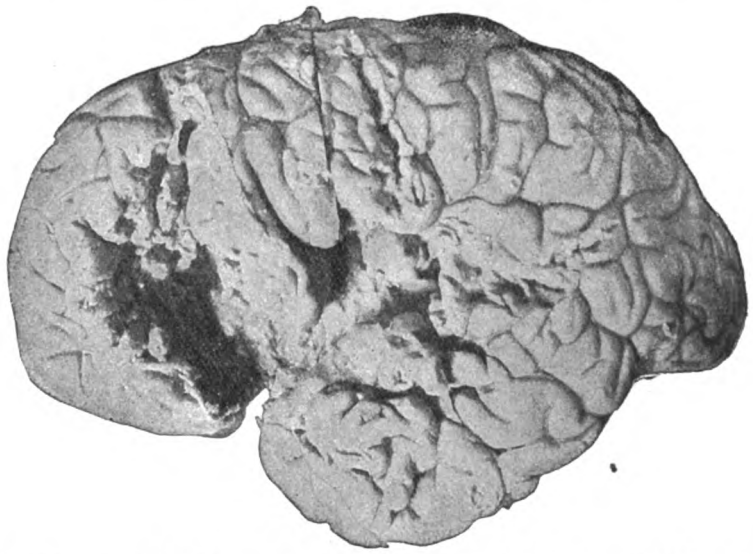

FIG. 345.—Photograph of a brain after total enucleation of a tumour about the size of a fist from the region of the left frontal lobe and the operculum of Arnold. Operation when moribund. Death from shock. The section was made post-mortem at the level of the Rolandic fissure. (Oppenheim.)

phining; but I regard palliative trephining as indicated, if the patient is suffering greatly from symptoms which cannot be otherwise relieved, or if the affection of his sight is increasing rapidly and threatening to develop into blindness.

In a few cases (Jolly, Wiener) in which a radical operation was impossible, opening of the skull has led the tumour to grow outwards through the opening made by the trephine, the symptoms being thus greatly relieved.

In superficial cavernous angioma, ligature of the vessels leading to it may cause the tumour to disappear, as Brush has observed (*Journ. Nerv. and Ment. Dis.*, 1904).

Cases have been described (Gould, Sidney, Ballance, Sick, Horsley) in which recovery followed opening of the skull, although the tumour was not found. It is impossible for us to determine what the error in

diagnosis can have been. Such a result would, we should think, be most likely to follow in serous meningitis. We must also remember the possibility of pseudo-cerebral tumour and of acute brain swelling, already discussed.

Puncture of the ventricles has also been recommended for alleviation of the symptoms, but so far no particular results of this method have been reported. Beck has again lately advocated it strongly. Finally, one may resort to the method of puncture through the intact skull, practised by Middeldorpf, Schmidt, and A. Kocher, and lately advocated by Neisser (see above).

Lumbar puncture may induce transient alleviation and improvement in a few cases. I have very seldom found lasting benefit. Heubner, Fraenkel, Goldscheider, etc., have now and again found it to be beneficial, and Wolff [1] in particular reports a case in which alleviation followed

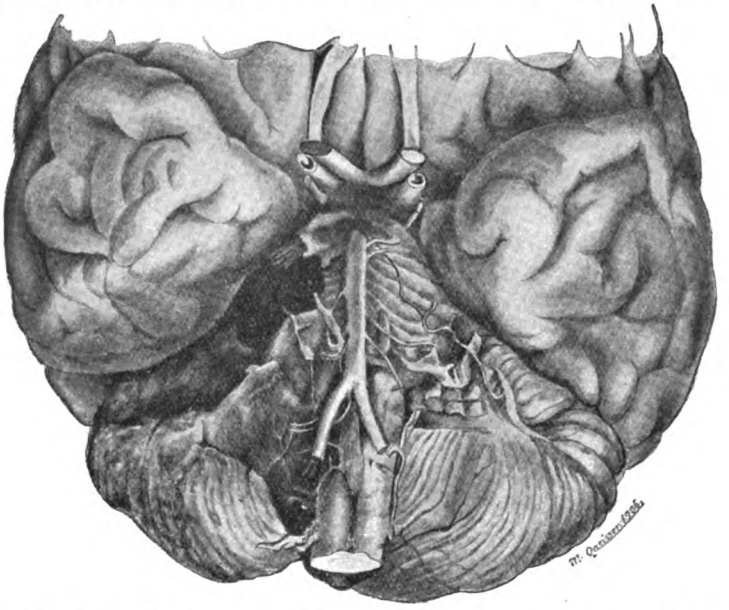

FIG. 346.—Appearance of the pons, etc., after successful enucleation of a tumour in the cerebello-pontine angle. (Oppenheim-Borchardt.)

each successive puncture, performed at intervals seventeen times. Widal also reports a case of this kind. G. Flatau has seen choked disc disappear in cerebellar tumour after lumbar puncture repeated seven times. But on the whole experience as to the therapeutic value of this measure in brain tumour is not encouraging. On the contrary, the troubles, especially the headache, have often been increased after puncture. I have seen it followed almost immediately by fresh symptoms of paralysis in a case of tumour of the corpora quadrigemina, in which only a little fluid could be obtained from the spinal canal. The tumour probably obstructed the Sylvian aqueduct. The operation has occasionally caused back and girdle pains, and, finally, it may endanger life, as is shown by the cases of Fürbringer, Lichtheim, Wilms, Stadel-

[1] *A. f. Kind.*, xxvi.

mann, Krönig, L. Müller, Finkelnburg, De la Camp, Gayet, Funkenstein, Masing, etc., and especially by the review by Gumprecht [1] (*cf.* also p. 767). It is therefore strictly necessary to carry out the puncture with great care (preferably by mean of Krönig's apparatus), with complete control of the pressure, to withdraw only a little fluid at a time, and to interrupt the operation immediately if the pressure is from the first very low, if it sinks with undue rapidity, or if the symptoms obviously increase. It should never be repeated if the communication does not appear to be free.

Surgeons differ very much in their opinions on the subject of treatment of bullets in the cranial cavity. Bergmann leaves them alone, as he has found that as a rule they become encapsulated and heal up, but he thinks that an operation for their removal is indicated when they have entered the motor region. Other surgeons, such as Henschen, Lennander, Burckhardt, Barker, Braatz, Chipault, Kümmel, Fredet, Drew, Laurić, Wieting-Effendi (*M. m. W.*, 1903), Périer (*R. n.*, 1903), Krause, and Holzknecht, relying on the definite local diagnosis made possible by radiography, have often removed bullets from the deep-lying parts of the brain. Bramaan and Wiemuth, and also Angerer, are practically of Bergmann's opinion. They regard trephining as permissible or imperative only (1) when the bullet lies on the surface, (2) when there is hæmorrhage from the middle meningeal artery, and (3) when there are marked symptoms of irritation of the motor region of the cortex.

Wilms (*Mitt. aus Grenzgeb.*, xi.) has noted the very interesting fact that lesion of the sympathetic plexus in the region of the cavernous sinus may give rise to a hyperalgesia in the area of the upper cervical segments—analogous to Head's experiments. He makes use of this symptom to locate the bullet. Milner (*B. k. W.*, 1904) and Vorschütz (*Z. f. Chir.*, Bd. lxxxviii.) have reported other cases of this kind.

Aneurism of the Cerebral Arteries

If we leave out of account miliary aneurism of the intracerebral arteries, and taken into consideration only aneurism in its ordinary form, we must certainly term it an exceedingly rare disease. On the other hand we should remember that it occurs more often in the vessels of the brain than in the arteries of other parts of the body. True aneurisms are not often found within the brain itself. They affect rather the *arteries of the base of the brain* before they or their branches enter the brain substance. Aneurisms may develop on each of these branches, the middle cerebral and the basilar artery being specially often involved (Lebert [2]). The points of bifurcation are favourite sites. The aneurisms are not infrequently multiple, as Morgagni has described. In addition to the sac-like bulging, there is a *general aneurismal dilatation* of the cerebral vessels, especially of the vertebral and basal arteries. The vascular tumour may be only the size of a pea or a bean, but in other cases it may be as large as a pigeon's or even a hen's egg. The arteries of the left side are more often affected than those of the right. The adjacent parts of the brain are not merely more or less severely compressed; they often show more or less extensive areas of atrophy or softening (Griesinger,[3] Leber, Oppenheim,[4] Monakow-Ladame [5]).

The formation of aneurisms is due to disease of the arterial walls, which destroys the contractile elements at circumscribed points or to a greater extent, their place being taken by fibrous tissue. The vessel

[1] *D. m. W.*, 1900. [2] *B. k. W.*, 1866. [3] *A. d. Heilk.*, 1862.
[4] *B. k. W.*, 1887, and Oppenheim-Siemerling, *Charité-Annalen*, xii.
[5] "Nouv. Icon de la Salpêtrière," xiii.

wall thus becomes distensile, and it may become greatly dilated by the weight of the blood column resting upon it. *Atheroma* and especially *specific arteritis* affect the vessel in the same way. We should specially keep in mind the fact that *embolism of the cerebral arteries* may give rise to the formation of aneurisms, the embolus causing on the one hand an incomplete occlusion of the vessel and on the other a local inflammation. The relation of *heart diseases* and *syphilis* is shown by the fact that aneurisms tend to occur during youth or middle age, and to be rare as a whole in advanced life. *Trauma* (head injuries) may give the impulse for their development. A few cases (Hofmann,[1] Karplus [2]) have suggested a *vasomotor* origin. In one of my patients an aneurism in the region of the deep cerebral artery developed immediately after a severe fit of coughing. In one lady under my care, who suffered from obstinate constipation, cerebral symptoms suddenly appeared, during straining, which could best be explained by the assumption of an aneurism.

Aneurism may give rise to *no symptoms* until the moment of *rupture*, which takes place in very many cases (48 out of 86, according to Lebert, in 147 out of 322 according to Hey [3]). It is revealed by symptoms of a cerebral hæmorrhage, which is usually slow and coming from a small opening, or it takes the form of an apoplexia ingravescens. It may in other cases give rise to very violent symptoms, *e.g.* a sudden fall to the ground, with loss of consciousness, deep coma, general convulsions, general paralysis, fever, asphyxia, etc. The rupture is not necessarily fatal; it may be repeated several times and thus produce paroxysms of severe symptoms (Kretz, Karplus). Intrameningeal hæmorrhage caused by rupture may be demonstrated by changes in the cerebro-spinal fluid found on lumbar puncture, and the diagnosis may be made in this way, as in the cases of Nothnagel,[4] Rindfleisch,[5] Ohm.[6] Penetration of the blood into the subarachnoid space may give rise to spinal symptoms, especially to those of meningeal irritation.

As a rule, however, the aneurism is revealed by the *general* and *local* symptoms of a brain disease.

The former include *headache*, often described as pulsating and sometimes resembling hemicrania (Fiedler,[7] Karplus), *vertigo*, *vomiting*, *stupor*, which may be present when the aneurism is a large one, and convulsions, which are rare. These symptoms are common to aneurism and to all the affections which contract the space within the cranial fossa. Choked disc is absent as a rule, but after rupture the blood may make its way into the sheath of the optic nerve and so give rise to ophthalmoscopic changes (H. White,[8] etc.). This kind of tumour is specially characterised by a *pulsating vascular murmur*, which may be heard over the whole skull or at certain sites, and often even at a distance. This is by no means absolutely pathognomonic of aneurism, as it may occur in tumours situated in the neighbourhood of a large vessel which they compress (Hennig, Jurasz,[9] Oppenheim, Bruns, etc.), and in very vascular tumours. It is especially not pathognomonic in childhood, as it may be

[1] *W. k. W.*, 1894.
[2] *Obersteiner*, viii.
[3] *Inaug.-Dissert.*, Berlin, 1898. See also Krey, *Inaug.-Dissert.*, Greifswald, 1891, and Vogel, *Inaug.-Dissert.*, Erlangen, 1895; Crouzon-Ficai, *R. n.*, 1907.
[4] *W. kl. W.*, 1902.
[5] *A. f. kl. Med.*, Bd. lxxxvi.
[6] *D. m. W.*, 1906.
[7] *Jahrb. d. Ges. f. Nat.*, Dresden, 1887.
[8] *Brit. Med. Journ.*, 1894.
[9] "Das systol. Hirngeräusch der Kinder," Heidelberg, 1877.

present in healthy children and in rickets, anæmia, hydrocephalus, etc. (Henning, Henoch, Roger, Jurasz, Strümpell, etc.). Even in adults it may be produced by anæmia and by vasomotor disorders. I have satisfied myself on this point in several female cases. A case by A. Fuchs shows that this symptom may also be due to idiopathic chronic hydrocephalus in the adult. It may possibly also be caused by congenital narrowness of the carotid foramen (Troeltsch, Urbantschitsch). Head murmurs have occasionally been noted in exophthalmic goitre, in pulsating exophthalmus, and in compression of the sympathetic nerve by tumours (Gowers). D'Allocco [1] diagnosed an aneurism practically from this symptom, and found instead merely an atheromatous degeneration of the cerebral arteries. This was also found in one of the cases described by Wallenberg. A vascular murmur due to an aneurism of the internal carotid and its branches can usually be checked by compression of the carotid at the neck.

The *local* symptoms are naturally dependent upon the site of the aneurism, and they point as a rule to a lesion at the base of the brain.

The paralytic symptoms may have a gradual onset, but they often develop in an acute, subacute, or relapsing manner. *Apoplectiform attacks* may occur during the course of the disease, and are due to the occurrence from time to time of marked increase in the size of the aneurism, to several repeated hæmorrhages, or to the displacement of one of the vascular branches arising from it.

Aneurism of the *internal carotid* may compress the optic, oculomotor, and olfactory nerves, and the first branch of the trigeminus. It thus gives rise to affections of vision, amblyopia, or the various forms of hemianopsia (Bramwell),[2] with or without ophthalmoscopic changes, oculo-motor paralysis, exophthalmus, pain, hyperæsthesia, and hypæsthesia in the region of the first branch of the trigeminus, and to anosmia. Neuroparalytic keratitis may also occur (Czermak). In a case described by Karplus, where the diagnosis was made during life, the paralysis affected the abducens and the external eye-muscles of the oculo-motor nerve. Pressure on the cavernous sinus may cause hyperæmia of the retinal veins (?) and dilatation of the veins of the face. If the tumour is a large one (situated on the left side) it may somewhat involve the speech centre, and by its distant action upon the motor conduction tract or by direct compression of the cerebral peduncle may give rise to hemiparetic symptoms. In rupture the blood may extend into the cavernous sinus and produce a pulsating exophthalmus.

It is a remarkable fact that aneurism of the carotid may cause erosion of the sella turcica (Bramwell). As regards arterio-venous aneurism of the internal carotid and the cavernous sinus, consult the text-books on surgery. Bruce and Drummond describe an aneurism of the anterior communicating artery (*R. of N.*, 1904).

Aneurism of the *arteries of the corpus callosum* is specially apt to involve the optic and olfactory nerves. In one case it is said there was unilateral temporal hemianopsia.

Aneurism of the *middle cerebral artery*, which very often remains latent until death, may also injure the oculomotor and olfactory nerves,

[1] *Rif. med.*, 1897.

[2] Intracranial Aneurisms, "Clin. Studies," 1906; *R. of N.*, 1906. See also as regards the ocular symptoms of aneurism of the internal carotid, Loeser, *A. f. Aug.*, Bd. l.

but it has a special effect upon the brain substance, and by means of compression produces aphasia, monoplegia, hemiplegia, etc. The paralysis is usually preceded by symptoms of motor irritation. Aneurism of the posterior communicating artery may, by compression of the optic tract, give rise to hemianopsia. Killian describes a case in which neuroparalytic keratitis, oculomotor paralysis, and blepharospasm were present. Hey, Karplus, and Rindfleisch have lately described aneurisms at this site.

In a case observed by Perazzolo (*Riv. di Patol.*, 1906) the disease produced no symptoms of any kind.

Aneurism of the *basilar artery* directs its pressure towards the pons, or perhaps towards the cerebral peduncle or the medulla oblongata. The pons may be deeply indented, the cranial nerves at this region may be dragged, crushed, and atrophied. The patient complains of headache in the occipital region, and the movements of the head are often restricted. Compression of the pons, etc., produces symptoms of *alternating hemiplegia* or *bulbar paralysis*, which develops subacutely or paroxysmally. The symptoms of irritation and paralysis are specially marked in the area of certain cranial nerves (v., vii., x., and xii., etc.).

Aneurism of the vertebral artery gives rise to similar symptoms (compare with chapter on acute and compression bulbar paralysis). The relapsing course is often very marked, as I have seen and Ladame and Monakow have described in one case.

In some of our cases in which the cranial nerves were alternately involved, *e.g.* the soft palate and vocal cord on one side, and the hypoglossus on the other, I have found aneurismal dilatation of the extremely tortuous arteries, so that the pressure was greater on different nerves of each side. We have also observed attacks of irregular action of the heart, dyspnœa, and rise of temperature, even above 40° C., lasting for some hours.

In aneurisms of the posterior cranial fossa, it has been several times observed that when the head was bent forward, severe disturbances of respiration occurred, the breathing becoming arrested. A relapsing course is to some extent characteristic of aneurism.

In one case (Massary and Carton, abstr. in *R. n.*, 1903) there were no bulbar symptoms of any kind, and the only sign of a basilar aneurism was a hemiplegia which developed and became complete in the course of fourteen days.

Aneurism situated near the origin of the *posterior cerebral* artery may cause alternate hemiplegia with involvement of the third and seventh cranial nerves (Rauchfuss, Delpech, etc.).

The *prognosis* of these aneurisms is very grave. In the great majority of cases death occurs within a few months or years. Spontaneous recovery has, however, been seen (due probably to the formation of coagula) (Hutchinson, Hodgson, Humble, Oppenheim).

Treatment in the majority of cases is purely symptomatic. Continuous use of iodide of potassium may induce recovery. In specific cases mercury is also to be recommended. No essential improvement can be expected from the use of ergot. Everything that increases the blood pressure in the brain or hinders the flow of blood must be avoided as far as possible. A light, non-stimulating diet should be ordered, alcohol, coffee, and tea being forbidden. Mild aperients are suitable. The patient's head should not lie low, nor be bent much forwards or backwards.

Aneurism of the *internal carotid* has been treated by ligature of the common carotid. This has been successful in a few cases (Cöe,[1] Lefort,[2] Parker), whilst in others it has proved fatal (Jeaffresson,[3] Karplus). Cazin thinks he has induced recovery by digital compression of the carotid.

Lumbar puncture is absolutely prohibited. In one case it was immediately fatal by causing rupture of the aneurism.

Parasites of the Brain[4]

Cysticercus cellulosæ often occurs in the brain. With the inspection of meat and the disappearance of the tænia solium, this disease is becoming gradually less common (Hirschberg). A great number of cysticerci are usually found; the whole surface of the brain may be covered and the brain substance studded with innumerable vesicles (Fig. 346). A single

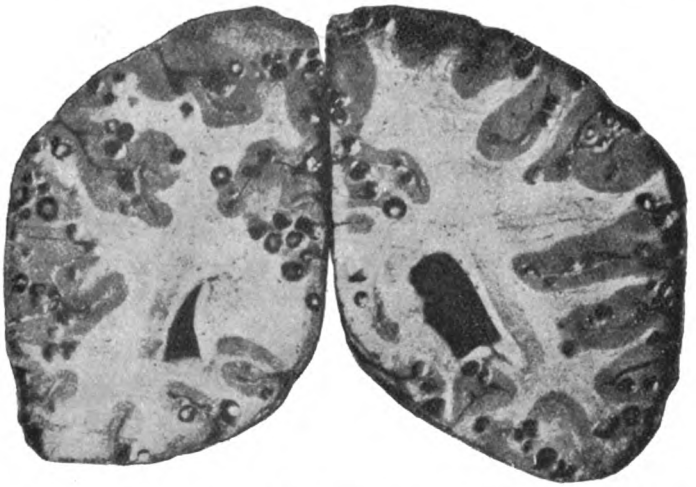

FIG. 347.—Invasion of the occipital lobe by cysticerci. (After Preobrashensky, reproduced by Jacobsohn.)

parasite is much less common. They are specially found in the meshes of the arachnoid and pia and in the bifurcations (*cf.* Figs. 348 and 349), less often deep in the medulla, in the ganglia, and rather frequently in the ventricles, swimming free or adhering to the ependyma (Fig. 350). They may also be seated upon the arteries (Zenker, Marchand, Askanazy, Monnier, Lévi-Lemoine[5]). They usually have a capsule of connective tissue. The secondary changes in the neighbourhood of the cysticerci are of great importance and often of great extent. In addition to the localised encephalitis and softening and to the small hæmorrhages in the vicinity, there may be periarteritis, obliterating endarteritis, and especially extensive *fibrous meningitis* (Zenker, Heller, Mennike, Askanazy,[6]

[1] *Gaz. Med.*, 1856. [2] In Epron, *Thèse de Paris*, 1890. [3] *Lancet*, 1879.

[4] Literature in Oppenheim, "Die Geschwülste d. Gehirns," 2nd edition, Vienna, 1902; Bruns, "Geschwülste d. Nervensyst.," 2nd edition, Berlin, 1908. Of the later works see Sato, *Z. f. N.*, Bd. xxvii.; Volovatz, *Thèse de Paris*, 1902; Wollenberg, *A. f. P.*, Bd. xl.; Henneberg, *Charité-Annalen*, xxx.; Stern, *Z. f. k. M.*, Bd. lxi.; Henneberg, *M. f. P.*, xx.; Jacobsohn, *M. f. P.*, xxi.

[5] *Nouv. Icon.*, xiv. [6] Ziegler's "Beitr.," vii.

Rosenblath [1]). Rosenblath has studied this cysticercus-meningitis with special care, and I have also examined two cases of this kind. In one of these, in which the process had extended to the cerebro-spinal meninges,

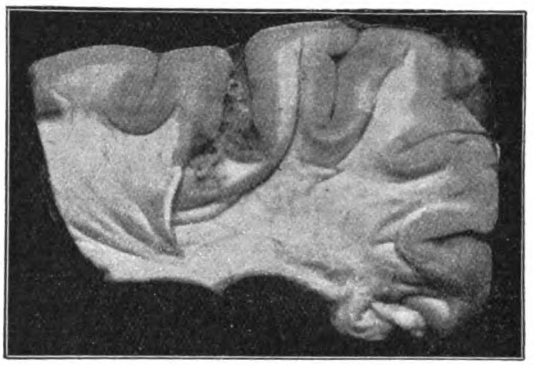

FIG. 348.—Cysticercus in a sulcus of the cerebrum. Natural size. (From a photograph of a hardened specimen in Oppenheim's collection.)

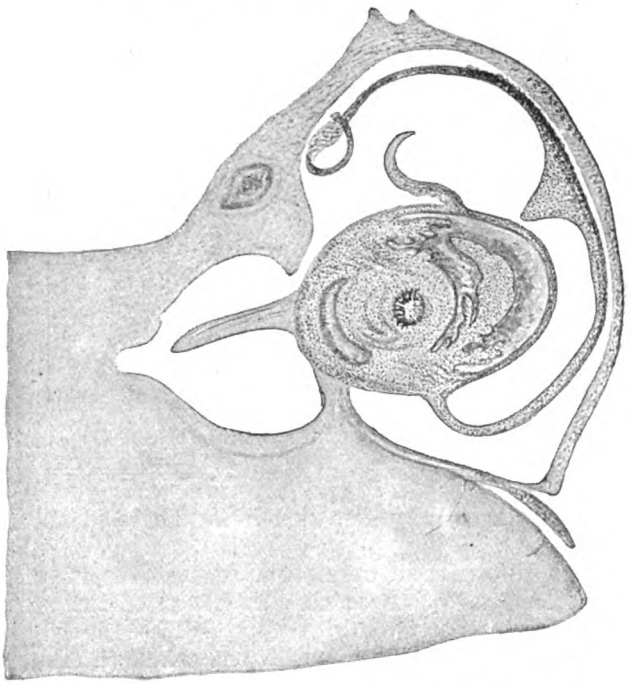

FIG. 349.—Cysticercus in the brain cortex. (From a microscopic specimen in Oppenheim's collection: under low power.)

I found the interpretation very difficult until I became conversant with his investigations. Henneberg [2] has also had an opportunity of investigating the pathological process in a number of cases, and he points out the great similarity of the meningeal and arteritic changes to those

[1] *Z. f. N.*, xxii. [2] *Charité-Annalen*, xxx., and *M. f. P.*, xx., Ergänz.

in gummatous basal meningitis. If the vesicle is situated in the ventricle, it may, by occluding the Sylvian aqueduct, give rise to marked hydrocephalus. Each tumour is as large as a pea or a walnut. The formation of daughter-vesicles may produce a branched, grape-like structure—*cysticercus racemosus*—which may attain a great size. It is situated preferably at the base and often in the region of the arteries of the circle of Willis. This is the form which is associated as a rule with cysticercus-meningitis.

In this vesicular tumour, close observation shows the head and neck of the worm standing out like a small, usually dark-coloured point, and microscopical examination shows it to have a crown of hooklets and four suctorial discs (Figs. 349 and 351).

Details as to the histological changes, especially of the capsule and its neighbourhood, will be found in Sato, Henneberg, and Jacobsohn (*M. f. P.*, xxi.).

Should the cysticercus die, the contents of the vesicle become calcified.

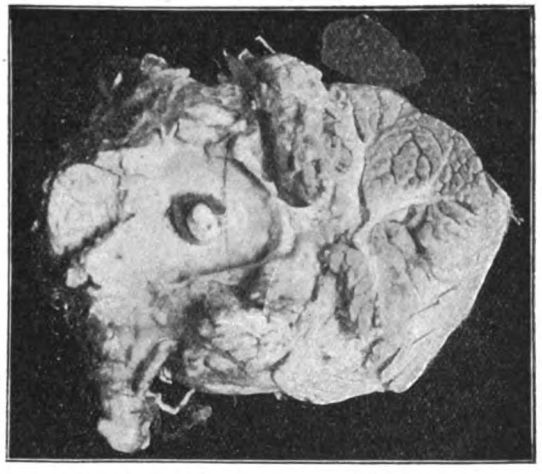

FIG. 350.—Cysticercus on the floor of the fourth ventricle in case diagnosed by Oppenheim. (Photograph of hardened specimen.)

Other regressive changes may also develop. There is no scolex in the sterile cysts.

There is great difference of opinion as regards the time at which the cyst dies. Stich speaks of three to six, Levin of ten to twelve, and Küchenmeister of fifteen to twenty years.

Hydatid cyst, a very rare disease on the whole (Cobbold found only 22 cases of hydatid cyst of the brain out of 327 cases which he collected), takes the form of a single or multiple tumour. A hundred vesicles have been found in one case. They are situated on the surface, both at the convexity and at the base of the brain, in its substance and sometimes in the ventricles. They vary greatly in size, from that of a pea to that of a man's fist. As a rule the vesicles are as large as a hen's egg.

Rieder (*Thèse de Paris*, 1904) gives some histological particulars.

Symptomatology.—Cysticercus is not unusually found *incidentally* during a post-mortem examination, especially if only one or two of its cysts are present. Even when it develops within the fourth ventricle, it may remain latent until shortly before death. A number of such cases are reported (Hammer, Meyer, Rothmann, etc.). Sudden death is not unusual in cysticercus of the fourth ventricle, and it may even occur without any warning when the cysticercus occupies the third ventricle (Kratter and Bonig).

As a rule, however, it produces a brain disease, with symptoms which vary greatly in their nature. They often do not point to any definite brain disease. Attacks of headache, an occasional fit of vertigo, transient

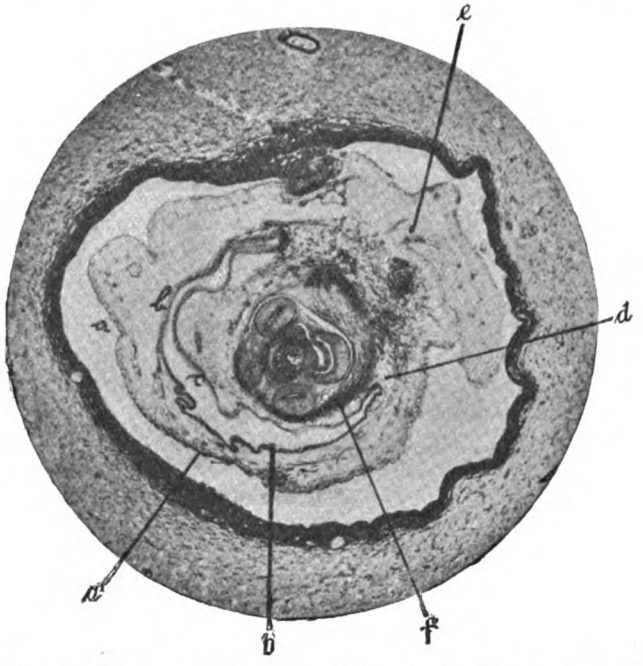

FIG. 351.—Cysticercus vesicle cut through the middle of its longitudinal axis. (a) outer, (b) inner envelope; (c) invaginated cysticercus; (d) and (e) loose tissue; (f) thick mass of fibres. (After Jacobsohn.)

confusion, and other symptoms of the same kind accompanied by vague subjective troubles suggest a diagnosis of hysteria, neurasthenia, hemicrania, vertigo, epilepsy, etc., until finally grave cerebral symptoms supervene, and raise the suspicion of a serious affection. In many cases these ominous symptoms are marked from the very beginning, but even in such cases there is as a rule no definite indication for the diagnosis of cysticercus.

Amongst the *symptoms* [1] of this disease, *convulsions* are a prominent feature (Griesinger).[2] The attacks sometimes correspond to *epilepsy*, more often to the type of *cortical* epilepsy, and more often still they are

[1] The descriptions given in earlier editions of this work have been confirmed by the more recent work of Sato, Wollenberg, Henneberg, etc.

[2] "Ges. Abhandlungen," i., 1872.

variable and *indefinite*. There is sometimes simple loss of consciousness, sometimes a condition of confusion. The mental disorder is usually accompanied by spasms in one extremity or only in one *group of muscles*, which *vary in site*, passing over from one side to the other. These spasms may persist for hours or days, or may be quite transient in nature. They are specially frequent in the *sterno-mastoid, trapezius, omohyoid and facial muscles*, etc. Transient tonic spasms, such as trismus, opisthotonus, and automatic movements may occur (Wollenberg). It is easy to see that these attacks, by their indefinite character and the frequent incompleteness of the loss of consciousness, may show a certain relationship to hysterical attacks, the more so as they may be preceded or followed by a general tremor, a fit of sobbing, or singultus. Roth and Ivanoff [1] rightly point out that the picture of the attacks may vary greatly even in each patient. According to Wernicke, cysticercus epilepsy is specially apt to pass into a fatal *status epilepticus*. It should also be noted that the epilepsy may be for a number of years, as many as twenty, the only sign of this brain disease (Hervé, Giannuli, Roth-Ivanoff, Pfeifer).

Henneberg's suggestion that the epilepsy has very often no connection with the disease, but that epileptics are infected with the eggs of the tæniæ only on account of their dementia, is applicable only to a few rare cases.

Mental impairment is a common symptom. Not a few of the cases reported came from asylums. There is usually simple *weakness of intellect*; sometimes there are *conditions of excitement*, hallucinatory delirium, states of *confusion* lasting for days and weeks, which, when combined with convulsions, may be diagnosed as epileptic equivalents, or when combined with weakness of the mind, may simulate paralytic dementia. The mental condition is also characterised by its variability and inconstancy. *Focal symptoms* often appear in the form of hemiparesis or hemiplegia, monoplegia, aphasia, etc. In a number of cases, the focal symptoms of the motor zone were very marked (Tietzen, Maydl, Posselt, Moltschanoff, Léon,[2] Fischer, Broca-Waquet,[3] etc.). Rosenblath found optic aphasia. F. Hartmann describes severe disturbances of orientation, impairment of stereoscopic vision, and a kind of mind-blindness. These symptoms are often of an inconstant nature. If the cysticercus lies in the fourth ventricle, it may give rise to *glycosuria, respiratory* and *circulatory disorders, vertigo, cerebellar ataxia, vomiting*, and symptoms of paralysis due to compression of the neighbouring structures. If the cerebellum is affected, as it is in not a few cases, occipital headache, vertigo, swaying, etc., are prominent symptoms. There is often a combination of symptoms arising from the cerebellum and medulla oblongata, as in cases reported by Oppenheim, Gerhardt, Czylharz, Long and Wiki, Bruns, Sato, Henneberg, etc. Vertigo and headache are among the most constant signs of the disease.

The *intermittent* character of these symptoms is very characteristic. Bruns (*N. C.*, 1902) has described an interesting case of cysticercus of the fourth ventricle, in which there was periodically violent headache, vomiting, and great vertigo, the patient being in comparatively good health between these attacks. He had merely to avoid turning his head quickly, as any sudden rotation to the left brought on vertigo and nausea, and might even cause him to fall down. Bruns

[1] *N. C.*, 1899. [2] *Thèse de Paris*, 1903. [3] *R. n.*, 1906.

regards this as a symptom pathognomonic of a free cysticercus in the fourth ventricle, and on this basis (and from the sudden death) he made a correct diagnosis in one case. I regard this Bruns' symptom, as I propose to call it, as a very valuable one, although I must admit that it occurs not only in free, but also in floating cysticercus of the fourth ventricle, and more rarely under other circumstances. This is admitted by Bruns himself (*N. C.*, 1906), but he maintains that the symptom in its well-defined form is characteristic of free cysticercus of the fourth ventricle. Osterwaldt and Stern agree with him, whilst Henneberg disputes the diagnostic value of the sign. The symptoms are probably chiefly caused by the internal hydrocephalus. In one case of cysticercus of the fourth ventricle in which I (*M. f. P.*, xviii.) had given this as the probable diagnosis, (see Fig. 350), the symptoms were characterised by attacks of vomiting and vertigo occurring at long intervals. During these attacks the patient had to abstain from taking any food, and he was obliged to avoid carefully any movement of his head. Headache was not always present, and it only became distressingly violent during the last attack. In the intervals the patient was so well that he was able to lead an ordinary life and to do his military service as an officer. His only inconvenience was that at times he had to avoid turning his head in any direction. The only permanent symptom was paralysis of the abducens or conjugate deviation and nystagmus.

Other cases of this kind have been lately communicated by Sato, Henneberg (*Charité-Annalen*, xxx., and *M. f. P.*, xx. Ergänz), Versé (*M. m. W.*, 1907), Osterwald (*N. C.*, 1906), Rautenberg (*D. m. W.*, 1905), and especially by Stern (*Z. f. kl. M.*, Bd. lxi.). In fifty-three out of the seventy-two cases which Stern collected, there was a *solitary* cysticercus of the fourth ventricle.

The cranial nerves are often involved. *Disorders of vision, deafness*, etc., have been repeatedly noted, although a marked choked disc has only been found in the minority of the cases. But it should not by any means be regarded as rare occurrence (Parinaud, Jacoby,[1] Wollenberg). In two of our cases there was slight transient optic neuritis, and in one transient choked disc. Marchand and Rosenblath have reported a case of sudden amaurosis. If the cysticercus is situated in the fourth ventricle or in the Sylvian aqueduct, the hydrocephalus to which it gives rise may produce simple amaurosis without any ophthalmoscopic change.

Symptoms of affection of the spinal cord or of the spinal meninges viz., hyperæsthesia (Wollenberg), Westphal's sign (Oppenheim), or intermittent absence, alternating with exaggeration, of the knee-jerk (Wollenberg), are occasionally present.

The symptoms point as a rule to a morbid process which has developed at several sites in the brain. The resulting symptoms of irritation and paralysis, the former especially, are not steadily progressive, but show remissions, the attacks being separated by intervals of complete good health. The condition shows therefore, on the one hand, some features of the neuroses and psychoses, and on the other some of brain tumour, especially in its specific form. In addition to these, there are symptoms due to the secondary changes, in particular to the hydrocephalus and meningitis. The clinical condition may thus very much resemble that of gummatous basal meningitis (Rosenblath). The power of movement possessed by the cysticercus has been thought to explain the inconstancy and variability of the symptoms (Merkel, Zenker, Kojewnikoff,[2] etc.). Marchand has attributed the rapid onset and disappearance of paralytic symptoms in cysticercus racemosus to changes in the fulness of the various vesicles caused by contraction of the parasite. Griesinger says that a brain disease in which paralysis is present from the first or from a very early period should certainly not be ascribed to cysticercus. Prolonged and progressive paralysis is particularly rare.

[1] *Kl. M. f. Aug.*, Bd. xli.

[2] *Korsakoff's Journal*, 1902.

The data given enable us here and there to give a *probable diagnosis*, as I, Bruns, Loewenthal, Osterwaldt, and others have successfully done. It is greatly strengthened by the presence of *cutaneous cysticerci*, but these are often not found in cases in which the parasites have made their way into the brain and internal organs. They are usually oval tumours, the size of a pea or a hazel-nut (seldom larger or smaller), lying *below the skin or in the muscles*. They are *movable*, and feel *firm like cartilage, tense and elastic*. They may increase to some extent in size from thickening of the capsule and increase of the fluid contents. The diagnosis can be made absolutely certain if these are excised and examined. The cysticercus sometimes appears in the *eye*, where it can be seen by the ophthalmoscope. Cysticercus of the eye, such as Hirschberg[1] describes, is, however, a very rare disease with us.

Another circumstance should be taken into consideration as regards the diagnosis, viz., whether there has been any *possibility of infection*, whether the patient has already suffered from a tape-worm (tænia solium) (which is, however, present in only a minority of the cases) or has lived with others who suffer from this parasite, or whether his occupation and mode of life, *e.g.* eating raw pork, suggest the possibility of an invasion by cysticerci. There is hardly any danger of the symptoms which are caused by the intestinal parasite itself being wrongly attributed to cysticercus of the brain. Peiper has, indeed, suggested that animal parasites in the human body produce poisons which are capable of causing the most varied cerebral symptoms, and cases of this kind have been reported by Marco, Festa, and others.

The presence of cutaneous cysticerci has repeatedly made it possible for myself and other physicians to explain correctly the brain symptoms which were present (Posselt[2] has lately described an interesting case of this kind; see also the thesis of Volovatz), whilst the cysticercus of the brain could be diagnosed only in very few of the cases under my observation in which these cutaneous cysticerci were absent, such as the one illustrated by Fig. 350. One patient, for instance, complained of headache and unsteady gait. She behaved like a hysteric. Whilst she could not take a step without staggering, she could walk straight forwards if she had a stick in her hand, although she did not lean upon it, or if one suggested to her in some other way that she was quite able to walk. At first it was thought that the discs were slightly dim, but this could not be confirmed later. In view of the entire absence of any objective symptoms and the peculiar mental condition of the patient, hysteria was given as the probable diagnosis, until one day she fell down unconscious and died. The autopsy revealed a large cysticercus in the roof of the fourth ventricle.

In another case a cysticercus of the fourth ventricle at first gave rise to paraplegia of all four extremities, which improved so far that only paralysis of the legs remained. In addition to this, mental confusion developed, with incontinence of urine and fæces. If the history and the mental condition had been disregarded, one would have had to give a diagnosis of cervical or disseminated myelitis. Moreover, it may be that the cysticercus involves both the brain and the spinal cord, so that the clinical condition may correspond with that of chronic cerebro-spinal meningitis.

[1] *B. k. W.*, 1904.

[2] *W. kl. W.*, 1899.

Hartmann (*W. kl. W.*, 1902) has shown that the diagnosis may be made by means of lumbar puncture, whilst Pfeifer (*Z. f. N.*, xxxiv.) has been able to establish it by means of puncture of the brain.

Sato's statistics, which show that the disease hardly ever occurs in youth, that it usually commences between the ages of 40 and 60, and very specially affects the male sex, are worthy of note.

The *prognosis* of cerebral cysticercus is always grave. *Arrest* and recovery are, however, not impossible, as calcified cysticerci, which have caused the patient no inconvenience in the last years of his life, have often been found in the brain. Henneberg and others have, it is true, denied that the disease is arrested by the death of the parasite. Some cases have been reported in which the symptoms of a brain disease improved, whilst a cysticercus became visible in the eye. I have treated one man for many years for cortical epilepsy, which from the presence of cysticercus in the skin had to be attributed to cerebral cysticercus. Eventually the attacks disappeared, and he may now be regarded as cured.

Treatment is mainly prophylactic. The use of raw or insufficiently cooked pork should be prohibited. A tænia should be expelled as soon as possible and rendered innocuous. The only other treatment open to us is symptomatic. It is indeed possible that a cysticercus of the motor zone, which gives rise to symptoms of a brain disease, might be successfully excised, but this treatment is usually a failure on account of the multiplicity of the parasites. In the second edition of this work I remarked that whilst this chapter was being written, a case of this kind had been reported from the Breslau surgical Clinic (Mikulicz-Tietzen) in which the cysticercus of the motor zone was found and removed; other symptoms, however, persisted and pointed to the multiplicity of the tumour. Maydl[1] has in the meantime described a very instructive case of this kind, in which the results of the operation were but transient, a second operation proving of no avail (Fischer).[2] F. Krause[3] also found the effect of operation to be very transient in its nature. Broca and Waquet[4] report "definite" recovery after operation. Pfeifer has communicated the remarkable fact that sensory aphasia has led to the local diagnosis of cysticercus of the left temporal lobe, which was confirmed by brain puncture and followed by operation. The result was disappointing on account of the multiple nature of the tumour. A case described by Krönig shows that lumbar puncture in cysticercus of the posterior cranial fossa or of the fourth ventricle constitutes a direct danger to life. On the other hand the presence of cysticercus vesicles in the cerebro-spinal fluid has confirmed, or greatly strengthened the diagnosis of cerebral cysticercus (Hartmann[5]). Henneberg does not consider that there is much danger in lumbar puncture, and he would expect it to have a favourable effect by diminishing the hydrocephalus. Care is specially necessary if the pressure diminishes suddenly.

Bruns has recommended puncture of the ventricle in cases where the diagnosis of cysticercus of the fourth ventricle is certain, and in which there is no indication that the vesicles are freely mobile.

[1] *W. kl. R.*, 1901.

[2] *M. f. P.*, xviii.

[3] Hirnchirurgie, "Die deutsche Klinik," etc., Berlin, 1904. Maragliano (*Gaz. d. Osped.*, 1904) has described the same case.

[4] *R. n.*, 1906.

[5] *W. kl. W.*, 1902.

I have previously expressed my disapproval of this procedure, but since I have seen F. Krause expose the region of the fourth ventricle in a living subject, and puncture the cavity without necessarily involving any danger to life, I am bound to recognise the correctness of Bruns' indication. I recommend, to begin with, that the patient be kept in bed for weeks, resting completely, and with his head fixed, with the object of facilitating a settlement of the freely mobile vesicles.

Hydatid cysts of the brain [1] may run their course without giving rise to any symptoms. As a rule symptoms of brain tumours are present. In one of my cases choked disc alone was absent, but it has often been noted as well as atrophy of the optic nerve and amaurosis. The headache is usually intensified by movement, and the patient has often the feeling that something is moving in his head (this sensation is, however, most often of mental origin, and is specially common in hypochondriac neurasthenia). Westphal [2] has noted a most interesting fact, viz., that the tumour may rupture outwards, through the bones of the skull and through the nasal cavity. The echinococcus erodes the bones, and forms palpable cavities within them, through which the fluctuating tumour makes its escape. A test puncture leads in such a case to a definite diagnosis of the echinococcus. Ninety hydatid sacs have been evacuated in this way, followed by recovery. A few similar cases have been reported in the literature (Reeb, Mudd, Clémenceaux, Castro, Morquio). Mudd thinks his patient was cured in this way. In a case reported by Esteves [3] perforation took place. On the other hand rupture of a hydatid sac may give rise to grave symptoms (brain symptoms, exanthema, etc.), due to auto-intoxication, and may thus prove fatal. In a case observed by Franke,[4] the thinning of the skull was only indicated by the cracked-pot sound (see p. 914), the skull also bulging considerably at certain points.

The number of cases in which the echinococcus produced symptoms of a tumour of the motor zone and gave occasion for an operation (Hammond, Fitzgerald, Esteves, etc.). has lately increased. Auvray collected from the literature sixteen cases, in which surgical measures had been adopted ; nine of these resulted in death and seven in recovery. A few additional cases of recovery have been reported, *e.g.* by Wiesinger and Saenger.[5] Esteves is very doubtful, however, as to the permanence of this recovery.

Focal symptoms arising from the temporal lobe have been observed by Sérieux and Mignot [6] in a case in which some of the vesicles had developed in this region of the brain.

The fact of echinococci being found at other parts (*e.g.* in the axilla in a case diagnosed by Sonnenburg) is of special value in deciding the diagnosis, but this does not often occur. Küchenmeister only found echinococcus in other organs in eleven out of eighty-eight cases of echinococcus cerebri.

Should this indication be absent, the diagnosis can only be a provisional one, as in the case described by Rennie and Crago, who rightly ascribed the signs of a tumour of the left frontal lobe to an echinococcus on account

[1] Comprehensive reviews are given by Morgan, Clémenceaux, Guérinau, Monsseaux, and Gothard-Riche (*Nouv. Icon.*, xiv.).

[2] *B. k. W.*, 1873. [3] *Semano Med. Buenos-Aires*, 1894, and *Prog. méd.*, 1899.

[4] *Z. f. Chir.*, Bd. lxvii. [5] *D. m. W.*, 1903. [6] *Nouv. Icon.*, xiv.

of local thinning of the bone and the probable chance of infection (the patient being occupied with dogs), and operated upon it with success. Franke and Loewenthal had also given this as the probable diagnosis. The case is usually simply diagnosed as a tumour or a chronic meningitis (*e.g.* by Gallichi). P. Jacob [1] arrived at the diagnosis in one case by lumbar puncture, the fluid containing hooklets, succinic acid, and large quantities of chloride of sodium. Repeated puncture is said to have had a beneficial effect in this case.

It may be mentioned also that an echinococcus embolism has been observed in the cerebral arteries; the vesicles probably originated from the left ventricle.

The occurence of *distomumcysts* in the brain is reported by some Japanese writers (Otani, Yamagiwa, Katzurada, and especially Taniguchi, under Jacobsohn's direction). The pathological and clinical conditions are very like those of cysticercus cerebri. See also the communication of Tsumoda-Shimamura (ref. *N. C.*, 1907) on katayama disease.

We cannot here discuss so-called *sleeping-sickness*, its origin and causes.

Hydrocephalus

Literature in Huguenin, Ziemssen's "Handbuch der spez. Path. u. Therapie." xi., 1878; in the textbooks on children's diseases by Rilliet-Barthez, Henoch, Baginsky, Finkelstein, etc.; also in D'Astros, "Les Hydrocéphalies," Paris, 1898; Schultze, Nothnagel's "Handbuch der spec. Path.," ix.; Weber, *A. f. P.*, Bd. xli.; Uhthoff, Graefe-Saemisch's "Handbuch," second edition, ii. T. xi.

For literature on acquired, acute, and chronic hydrocephalus, see further on.

In the majority of cases hydrocephalus is a *congenital* disease. But it may be *acquired* in childhood, in youth, and even during adult life. It seems to us expedient to discuss separately the congenital and the acquired forms.

As yet we do not understand the causes and the origin of *congenital hydrocephalus*. It has been assumed that trauma, or mental excitement affecting the pregnant woman, may be a cause, but the influence of these factors is still hypothetical. Cachexia, intoxication, and especially syphilis are with more reason regarded as causes. Cases of the latter kind have been reported by Bärensprung, Sandoz, D'Astros,[2] Heller,[3] Hochsinger, Neumann, Solovtzoff, Andéoud,[4] and in particular by Fournier. I have in a few hereditary syphilitics found a moderate degree of hydrocephalus, which at the age of puberty and later gave rise to marked symptoms. Acute infective diseases of the mother during pregnancy may have the same effect (Gabail). It is also certain that family disposition plays a definite part. There are some families in which several children have suffered from hydrocephalus, the descendants of the the other members of the family having been born with this disease.

Congenital hydrocephalus consists of the accumulation of an excessive quantity of fluid in the cerebral ventricles. We have no definite knowledge as to its pathogenesis. It is supposed that the cause is *an inflammation of the ventricular ependyma* of the transudation. Others assume that there is *displacement of the communicating orifices* which connect the

[1] *Fortschr. d. Med.*, 1903.
[2] *D. m. W.*, 1892.
[3] *Rev. mens. des malad. de l'enfance*, 1891.
[4] *Rev. méd. de la Suisse rom.*, 1899.

ventricles with each other and the ventricular spaces with the subarachnoid region. There is no doubt that hydrocephalus may be due to this cause, but we do not know when and how frequently this factor is concerned. It is assumed by D'Astros, Boenninghaus,[1] Dexler, and Weber to be of great importance in the production of hydrocephalus. Pathological changes of this kind have been described by Luschka, Monro, Neurath,[2] Bourneville et Noir[3] (whose case is possibly one of acquired hydrocephalus), Spiller,[4] and others. Huguenin would distinguish between an inflammatory and a dilatation-hydrocephalus, the primary factor in the latter being the flexibility of the walls of the skull. We may pass over the other theories (hypoplasia of the suprarenal capsules, etc.) without discussing them.

We may mention teratoma of the brain as an extremely rare cause of congenital hydrocephalus, as in the case of Hulst, *Beitr. z. Geburtsh.*, viii.

We speak of an *internal* and an *external* hydrocephalus, according to whether the fluid fills the ventricle or the subarachnoid space. The latter form is much less important, and is usually simply a condition secondary to other brain diseases (hydrops evacuo due to brain atrophy, etc.).

The fluid, which is generally clear and colourless, contains little albumen and a small amount of salts (NaCl, etc.). It dilates all the ventricles, or it may be the lateral ventricle in particular. If the communications between the ventricles are displaced, the hydrocephalus may be limited to single ventricles (see below). The fourth ventricle is the least affected as a rule, but in exceptional cases the accumulation of fluid may be chiefly localised in it. The quantity of fluid contained in the cerebral cavities varies greatly—amounting from a few ounces to several litres (ten, twelve, and more). On an average there is about one litre of fluid.

The development of the brain substance is almost always impaired. It is more or less markedly reduced, to such a degree even that the wall of the hemisphere may be merely a layer of several millimetres in thickness. The convolutions and sulci are entirely obliterated, whilst the brain mantle encloses a thin, fluctuating sac of fluid. The central ganglia are often markedly flattened and the floor of the third ventricle bulges outwards like a cyst. There was an extraordinary inhibition of development in a case examined by Tuczek-Cramer (*A. f. P.*, xx.), and another by Homén, and still more marked changes (aplasia of the whole brain more or less) are described by Durante and Solovtzoff. The ependyma of the ventricles seems to be usually granular, and inflammatory changes are sometimes found in the choroid plexus.

Engel (*A. f. Kind.*, 1905) mentions hæmorrhage as a complication of congenital hydrocephalus.

The *skull* is always *increased in size*. In the normal new-born infant it is 35 to 40 cm. in circumference, and during the first year it grows to 45 cm., whilst in hydrocephalus it reaches 60—80—100 cm., and in one case it was even 167 cm. round. The skull is *rounded*, and the *frontal*

[1] "Die Meningitis serosa acuta," Wiesbaden, 1897.
[2] *W. m. Presse*, 1895.
[3] *Prog. méd.*, 1900.
[4] *Amer. Journ. Med. Sc.*, 1902, and *Journ. Amer. Med. Assoc.*, 1907.

and parietal protuberances are usually very prominent. The orbital roof is pushed downwards (Figs. 352, 353, and 354). The skull bones are usually thin ; they may be as thin as paper. The sutures and fontanelles are dilated. The latter remain open for an abnormal length of time ; they have in a few casse been found open in the third decade of life.

Symptoms and Course.—If the hydrocephalus has fully developed at the time of birth, it may effectually prevent delivery, and thus not a few hydrocephalic subjects die during birth. In most cases the hydrocephalus only becomes completely developed after birth. The skull, which at first is not materially enlarged, shows a considerable increase in

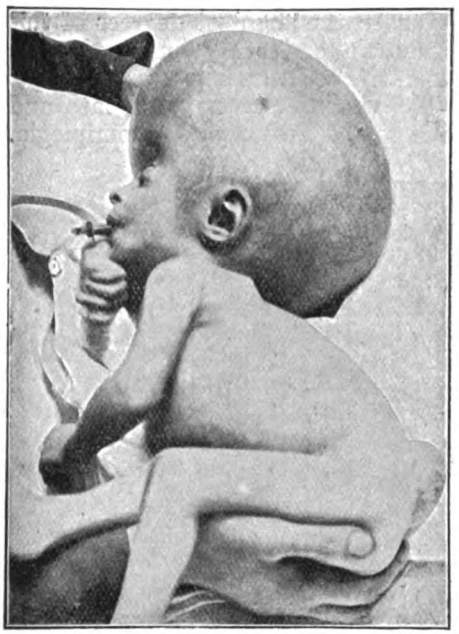

FIG. 352.—Condition of skull ; position of eyes, etc., in hydrocephalus. Combination with spina bifida. (Case of Oppenheim's from Bergmann's Clinique.)

FIG. 353.—Hydrocephalus. Intelligence unimpaired. (Oppenheim.)

circumference during the first weeks and months, as may be ascertained by measurements taken at short intervals. It may increase 1 cm. in a week and more.

The characteristic signs of this disease are *changes in the size of the skull, in its constitution and form*, and on the other hand, *anomalies in the function of the brain*. As regards the former, the symptoms due to increase in the size have already been described. The disproportion between the size of the skull and that of the face is particularly remarkable. The increase in the size of the former is sometimes specially evident in the sagittal diameter ; the skull is markedly dolichocephalic. The eyes look downwards. The veins are usually very prominent. In one of our cases the frontal veins formed thick blue cords, which could be traced from the root of the nose over the whole of the forehead. The hair of the head is usually very scanty. The thinness of the skull bones can often be recognised by palpation ; they may even be so transparent that when the skull is illuminated the vessels shine through them. A loud vascular murmur is sometimes heard on auscultation. The fontanelles are wide and prominent, the sutures gaping open.

The most important and constant cerebral symptom is the defective development of the intelligence. The majority of the children who suffer from this disease are *idiots* or *imbeciles*; they either cannot learn to speak at all, or they do so late and incompletely. Only five out of forty-one hydrocephalics could attend school (Wyss). It may happen, however, that the intelligence is only moderately affected, and not a few cases have been known in which the mental power was normal (as in the case illustrated by Fig. 353). Indeed the slightest degrees of this disease have been observed in individuals eminently endowed with mental, and especially with artistic talent (Perls). The *motor functions* are usually more or less impaired. The child learns to walk late and awkwardly, if he learns at all. There is sometimes complete hemiplegia.

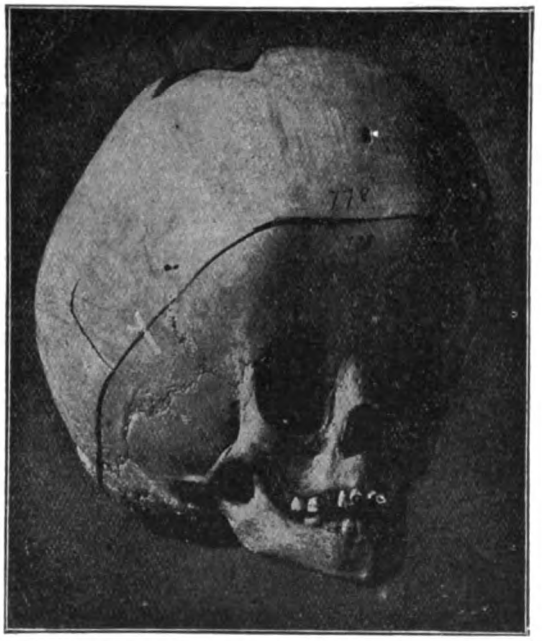

FIG. 354.—Hydrocephalic skull. (After Pfaundler-Zappert.)

The trunk is contracted, the movements of the arms are awkward, unsteady, and feeble, and the head is bent forwards, partly on account of its weight. *Spastic symptoms* are often present in the legs. The incontinence of urine and fæces so often present is mostly due to the mental condition, but it may also be due to paralysis. In many cases there are changes in the fundus of the eye, viz., *choked disc* or *neuritic atrophy*, but this symptom is more frequently present in the acquired form of hydrocephalus. Symptoms of paralysis in the other cranial nerves and sensory disorders are rarely met with in congenital hydrocephalus. General spasms of an epileptiform character are not uncommon. Feverish attacks with stupor, vomiting, etc., sometimes occur in the course of the disease.

Premature appearance of the *menses* and of *puberty* has been observed by Bourneville-Noir.

The hydrocephalus is often associated with other malformations, such as spina bifida (Figs. 352 and 205), hare-lip, encephalocele, club-foot, dwarfism, etc. I have seen it combined with albinism.

The affected individuals usually die soon after birth, mostly during the first few months or years of life. The exceptions to this rule are, however, not uncommon, and there is hardly any age which may not be attained in individual cases. One patient even reached the age of seventy.

It was recognised by the earlier writers (Magendie, Willis) and specially emphasised by Huguenin, that the cerebro-spinal fluid might sometimes make its way outwards, most often through the ethmoid bone towards the nasal cavity, the cerebro-spinal fluid thus escaping from time to time, or in a continuous flow (hydrorrhœa nasalis). This symptom is not rare in acquired hydrocephalus, and will be further considered under that heading.

In a few rare cases spontaneous perforation of the cerebral membranes and an escape of fluid through the sutures has been observed. Huguenin has collected nine cases of perforation, five of which were said to recover. Erosion of the orbital roof and the formation of orbital brain hernia was described by Buchsbaum (*W. kl. W.*, 1902). Further, injury of the skull bones may give rise to evacuation of the cerebral fluid, but it may also entail danger to the brain (Markwald).

As regards the *differential diagnosis*, special care must be taken to avoid confusion between the hydrocephalic and the rickety skull. The latter is squarer, and more like a box ; the fontanelles are not prominent, and other signs of rickets are present, whilst the brain symptoms are absent. Rickets may of course cause hydrocephalus to develop. There are also some healthy persons who have very thick skull bones.

Hyperostosis of the base of the skull in rickets (Chiari, Regnault, Homén, etc.) is usually associated with hypoplasia of the brain, and may thus give rise to confusion.

Further, there are *peculiar malformations of the skull*, either congenital or occurring during labour (see p. 714), which may be combined with neuritis or atrophy of the optic nerves, the skull as a whole not being increased in circumference. There is one form in particular in which the basal zone of the skull, especially its anterior part, is enlarged, so that the malar bones are very prominent whilst the skull is otherwise rather diminished in diameter (tower-skull). I have seen the veins of the face stand out very prominently in such cases. The intelligence may be quite normal. I am unable to say whether in such cases hydrocephalus is present and somewhat masked by the other malformations of the bones of the skull, but I have found complete absence of increase of pressure, or increase in the amount of cerebro-spinal fluid on lumbar puncture The paper by Oberwarth, *A. f. Kind.*, Bd. xlii., and that by Uhthoff may be consulted. The affection of the optic nerve is said to be a result of the hyperostosis of the bones of the base of the skull and the narrowing of the optic foramen to which it gives rise. In a case under my observation the deformity of the skull was undoubtedly the result of injury during birth, caused by contraction of the pelvis.

Hydrocephalus may undoubtedly be associated with microcephaly (hydro-microcephaly).

Hypertrophy of the brain (Virchow, Obersteiner, Brouardel, Tsiminakis, Variot, Schick), a very rare condition, is clinically allied to hydrocephalus, but has no connection with it as regards its anatomo-pathological nature. In a case of this kind described by Anton (*W. kl. W.*, 1902), the brain weighed 2055 grammes (about 72·5 oz.). There was a persistent thymus, and the supra-renal capsules were degenerated. Marburg (*Obersteiner*, xiii.) has lately published a careful study of this condition.

The affection described by Scheuthauer (*Allg. Wien. m. Zeit.*, 1871) as "a combination of rudimentary clavicles with anomalies of the skull," and by Marie-Sainton (*Bull. de la Soc. méd. des hôp. de Paris*," 1897 and 1898) as *dysostose cléido-cranienne héréditaire*, is distinguished from

hydrocephalus by the rudimentary development of the clavicles. See also the paper by Fuchs, *W. kl. W.*, 1907

The *prognosis* of congenital hydrocephalus is very grave. The possibility that life may be preserved and that the development of grave symptoms may not occur only applies to cases in which the disease is very slight in degree. When it is severe, death always occurs early and the mental functions are seriously affected. Spontaneous external rupture so seldom takes place that one can hardly reckon with it as a possibility. The results of treatment are also so uncertain that even taking them into consideration the prognosis must be regarded as unfavourable. D'Astros and Raczynski have expressed particularly pessimistic views as to the prognosis.

Treatment.—The aim of treatment has been to withdraw so much water from the organism that the loss of it will lead to absorption of the fluid contained in the cavities of the brain. For this purpose *derivatives* and *diuretics* have been prescribed. Experience has so far shown that this method is almost of no avail. Success has been reported in a few cases by Heller, Andéoud, Neumann, etc., from the use of *iodide of potassium* and still more of *mercury* (internal use of calomel, baths of sublimate or treatment by inunction). This treatment is said to have been successful in a very few cases, even when not syphilitic (Immerwol,[1] etc.).

Another mode of treatment is to counteract further enlargement *by compression* of the skull. No definite results have been obtained in this way and forcible compression must in itself be regarded as dangerous. Trousseau recommends for this purpose compression by means of strips of adhesive plaster, applied from each mastoid process to the external part of the other orbit, from the root of the nose to the external occipital protuberance in the direction of the sagittal suture, and then in circles round the head. Others use a broad elastic band.

Surgical treatment of congenital hydrocephalus has been successful in a very few cases. Puncture of the ventricle, which was recommended by Hippocrates and has since been often tried, is usually a failure. The danger of this treatment has certainly been greatly diminished by the use of stringent antisepsis, but still many cases end fatally. According to the statistics of Henschen,[2] based on 63 cases, recovery followed in 15, improvement in 12 cases, whilst 12 were treated with no result, and 24 died from the effects of the operation. Puncture followed by drainage of the ventricle cavities, such as Bergmann, Keen,[3] Kocher, Broca,[4] Robson [5] and Watson Cheyne [6] adopted, is particularly dangerous, as 20 out of 23 individuals treated in this way died. This is the case also as regards the injection of iodine, although it has been reported to be successful in a few cases. There is great difference of opinion among surgeons as to the value of operative treatment and the indications for it. Some think that it should only be resorted to in order to save life in the most severe cases; others regard it as contra-indicated only in the most and the least severe cases, whilst in all the others they would admit operation, either as a palliative or a curative measure. They advise, for instance, that no operation should be performed if the greatest circumference of the skull does not exceed 60 cm. in a child of six to eight months. In

[1] *A. f. Kind.*, xxxii.
[2] Penzoldt-Stintzings "Handbuch," xv.
[3] "X. Intern. Kongr.," Berlin, 1891.
[4] *Rev. de Chir.*, 1891.
[5] *Brit. Med. Journ.*, 1890.
[6] "Pediatrics," 1899.

any case it is necessary to use the most stringent antisepsis, to withdraw only a small quantity, keeping the pulse, respiration, etc., under observation, and to follow the operation up by the application of a compression bandage (Huguenin). Henschen thinks operation is justified when the pressure upon the brain is steadily increasing, when either life or reason is in danger, etc., and contra-indicated when the hydrocephalus is stationary and the sutures of the skull are closed by ossification. I have treated a woman of 48, who had undergone puncture in her childhood with such excellent results that one might speak of her as cured.

Quincke has recommended puncture of the spinal-canal (see p. 762), which has since been carried out by Bergmann, Soltmann, Kohts, Leyden, Repetta, Ranke, Raczinski, Oppenheim, Chipault, and others. It presents less danger, certainly, but is quite useless in cases in which there is no communication between the cavities of the skull or brain and the spinal cord, and it is very seldom helpful in other cases. Aspiration should be absolutely avoided. The decrease in the pressure should be controlled by the manometer or by means of Krönig's capillary tubes. It should not take place too rapidly or exceed a certain limit. It is much wiser to withdraw small quantities of fluid repeatedly at long intervals, a method which Grober [1] thinks has led to complete recovery in one case. Knöpfelmacher has also had remarkable success in a case in which he made sixty-six punctures, usually withdrawing 10 to 30 c.cm. each time.

Other attempts have been made to imitate the process of natural recovery and to select a mode of operation by which the fluid will ooze out constantly but very slowly. Ventricle drainage by means of a horse hair was used for this purpose. Quincke tried subcutaneous slitting of the spinal dural sac, but without any practical success. What is required, as Henle says (*Mitt. aus d. Grenzgeb.*, i.), is to establish a connection through which the fluid can pass between the ventricle and the surface of the brain or the subcutaneous tissue. For this purpose he uses a glass-wool nail which becomes encapsulated. The result of this treatment was very satisfactory in one case.

Similar methods have been adopted by Mikulicz, Sutherland, and Watson Cheyne (*Pediatrics*, 1899), Senn (*Alienist*, 1903), and Taylor (*Amer. Med. Journ.*, 1904), but so far without any essential success. Hoeven (*W. kl. R.*, 1904) has published a successful case. Many surgeons have objected to the method. Surgical text-books should be consulted with regard to its technique. Payr (*D. m. W.*, 1906) made use of a piece of the saphenous vein of the patient, with which he says he connected the ventricle with the sinus. Krause reports another method (*B. k. W.*, 1908).

The method of external *derivation* applied to the skull, which has occasionally been carried so far that the skin of the head, soaked with spirits of turpentine, has caught fire, has again recently been advocated.

Acquired Hydrocephalus

Literature in Billroth, *W. med. Bl.*, 1869; Seitz, "Der Hydroc. acut. d. Erwachs.," Zürich, 1872; Huguenin; Schultze, *loc. cit.*; Leber, *Arch. f. Ophth.*, 1883; Hogg, *Med. Press*, 1888; Oppenheim, *Charité-Annalen*, xv., and *M. f. P.*, xviii.; Quincke, "Volkm. Samml.," *N. F.*, 1893; *Ibid.*, *Z. f. N.*, ix.; *Ibid.*, *D. m. W.*, 1905; Boeninnghaus, "Die Meningitis serosa acuta," Wiesbaden, 1897; Krönig, "Verhandl. d. Kongr. f. inn. Med.," 1899; D'Astros, "Les Hydrocéphalies," Paris, 1898; Degré, *W. m. W.*, 1903; Weber, *A. f. P.*, Bd. xli.; Riebold, *D. m. W.*, 1906.

See also Uhthoff, *loc. cit.*, and compare literature in chapter on meningitis.

The cause and nature of this affection are still in many respects obscure. It is certainly the case that a hydrocephalus which is slight at birth, and possibly latent and unnoticed, may at any period of childhood,

[1] *M. m. W.*, 1900.

youth, or even of adult life become aggravated, either spontaneously or as the result of an injury, sunstroke, etc., and may by rapid and marked increase of the ventricular exudation give rise to serious symptoms. But there are many cases in which this cannot be assumed to be the mode of origin. Let us first consider separately the forms in which the hydrocephalus is of purely *secondary* importance and of no practical clinical interest. These include the cases due to obstruction which is caused by the pressure of a tumour upon the great vein of Galen, and the prevention of the return flow through the veins from the interior of the skull. Compression or obstruction of the aqueduct of Sylvius may also prevent free communication between the ventricles, and may in this way produce hydrocephalus. Thus many cases have been reported in which a cysticercus in the Sylvian aqueduct has obstructed the tract between the ventricles and so given rise to hydrocephalus. In other cases the cause has been occlusion of the fourth ventricle by a cicatrix, marked proliferation of the ependyma within it (Spiller), or tuberculous inflammatory closure of the entrance to the inferior cornu (Cramer-Weber). It is well known that the suppurative and in particular the tubercular form of meningitis are often accompanied by hydrocephalus. Special attention, however, should be given to the fact that hydrocephalus may be the permanent result of an epidemic (and sporadic) cerebro-spinal meningitis (Finkelstein, Zuppinger, Koch, etc.).

Thus Sorgente (*Pediatrics*, 1905) has found the meningococcus intracellularis in the fluid in several cases of hydrocephalus. Weber has further shown that diffuse and focal disease of the brain in the region of the ventricle may give rise to the development of hydrocephalus which, under these conditions, may be limited to one side or to one ventricle. These specially include localised atrophy, softening, and sclerosis due to atheroma of the vessels or cystic degeneration, etc., in the region of the walls of the ventricle.

The forms which appear in marasmus, phthisis, nephritis, etc., should not be regarded as independent affections.

External hydrocephalus is generally of *secondary* importance. It is the common result of cortical atrophy, whether of senile or other origin. It may also follow or accompany meningitic or encephalitic processes. There is, however, a primary external hydrocephalus, the symptoms of which are practically analogous with those of internal hydrocephalus (see below).

Then we have another form of "*idiopathic hydrocephalus*," as to the genesis of which many theories have been suggested. It may occur at any time of life, but is rare in old age, whilst childhood, even on physiological grounds, seems to be specially predisposed to it (Mya). The most probable explanation is that the hydrocephalus in these cases is a secondary process due to a *simple basal meningitis*, which has adhesions that have closed the communications (foramen of Magendie) between the ventricles and the subarachnoid space. This view accords with the facts in some cases, but in others there is no sign of this meningitis. I have described one such case in 1889, and on its evidence I must adhere to my belief in the existence of a *primary idiopathic hydrocephalus*, and I would lay special emphasis upon its clinical relationship to cerebral tumour. Annuske [1] had previously reported a similar case, and others were subsequently described by Eichhorst,[2] Kupferberg,[3] etc. Quincke has since

[1] *Arch. f. Ophth.*, xix. [2] *Z. f. kl. M.*, xix. [3] *Z. f. N.*, iv.

then directed his attention to this subject, and he maintains the view that a *serous meningitis of the ventricles*, a simple serous inflammation of the intracerebral pia—of non-parasitic nature, analogous, he thinks, to serous pleuritis—is in not a few cases the cause of an internal hydrocephalus, which makes its appearance in childhood or later, and may have an acute or chronic course. The exudation in such cases arises mainly from the choroid plexus. The pia and covering of the cortex may also be the site of simple inflammation and may produce external hydrocephalus. Trauma, mental exhaustion or excitement (Nonne), alcoholism, and acute infective diseases (pneumonia and typhoid in particular) are regarded as the *causes* of this disease. The importance of infective processes has been pointed out by Haushalter-Thiry, Leroux-Conzetti, Münzer,[1] Mya,[2] Netter, Parkes-Weber, Patel, and others. Parkes-Weber agrees with Quincke in thinking that this serous meningitis is analogous to serous exudation into the pleura and peritoneum. Münzer, on the ground of Quincke's reports and his own experience, thinks that is a very prominent factor in the etiology of tuberculosis. Biedert is also of opinion that a serous meningitis may develop from tuberculosis; Riebold agrees with him, and Heubner does not deny the possibility. Quincke's theory of a non-parasitic origin of this meningitis cannot therefore be entirely maintained (Münzer), as bacteria (staphylococci, typhoid-bacilli, etc.) have sometimes been found in such cases, although this would certainly appear to be the exception. Quincke himself admits in his latest work that a non-suppurative exudation may develop owing to the fact that the virulence is very slight and the microbes few in number. I have seen the disease occur in the puerperium and in association with nephritis. The diagnosis which I made in this case was confirmed by autopsy. Other cases (Levi, Joel, Kretschmann, Oppenheim, Lucae, Waldvogel, Hammerschlag, R. Müller, Brieger, Hegener, Hansen, Broca, Lecené-Bourgeois,[3] etc.) have shown that this form of meningitis is not uncommon in purulent otitis, and that it may arise from the nose and its accessory sinuses (Herzfeld [4]). It has also been attributed to toxæmia (Seitz). Many writers attach great importance to the bacterium coli. Mya also emphasises the toxic origin of acute hydrocephalus.

If the affection develops acutely, it is difficult to distinguish it from suppurative meningitis, and still more from tubercular meningitis. There is usually, however, no rise of temperature, or it is slight and inconstant. If fever is present at the beginning, it rapidly passes off. The headache is also less severe and the rigidity of the neck less marked. In a case described by Seiffer,[5] it is true, the fever, for a time at least, was pretty high, and the muscular rigidity was also marked. Riebold [6] has occasionally observed this condition. As a rule the mental confusion is not permanent, but recurs from time to time. Sight is usually very much affected. Finkelnburg [7] describes very slow pulse and Cheyne-Stokes respiration in one case. The former symptom has been observed by Nonne and myself. This form of meningitis may also give rise to spinal symptoms, such as Westphal's sign, which Burr and M'Carthy found to be inconstant. Goldscheider has seen the knee-jerk return in one case after

[1] *Therap. med. Woch.*, 1899.
[2] *La Settim. med.*, li.
[3] See the corresponding data on p. 760 and in Körner, *loc cit.*
[4] *B. k. W.*, 1905.
[5] *Charité-Annalen.*, xxiv.
[6] *D. m. W.*, 1906.
[7] *M. m. W.*, 1904.

lumbar puncture. Herpes is hardly ever present; Henschen alone says he has observed it in a case of this kind.

Lumbar puncture is a valuable aid to the recognition and differentiation of this condition, as in serous meningitis if the channels by which the cerebro-spinal fluid can pass from the brain to the cord are not occluded, the fluid obtained is clear, with very few cells, and is subject to high pressure.

A definite diagnosis is often impossible until a late stage of the disease, as this *acute acquired hydrocephalus*, or acute serous meningitis, may terminate after a few weeks or months in complete or incomplete recovery. In other cases it develops into a chronic condition, in which the symptoms (headache, vertigo, vomiting, optic neuritis, cerebellar ataxia, etc.) which correspond exactly to the chronic form already described, or some ocular affection alone (atrophy of the optic nerve, blindness) point to the previous existence of the brain disease. This class undoubtedly includes some of those not uncommon cases in which the patient develops symptoms of a severe brain disease, which completely disappear within a few weeks or months, leaving only permanent blindness, usually associated with atrophy of the optic nerve secondary to neuritis. Quincke reports several cases of this kind, in which the cardinal symptoms were headache, vomiting, stupor, and optic neuritis, a remittent course of several weeks or months terminating in recovery (under treatment by mercury). I have frequently seen such cases. An intermittent course is not unusual, and a fatal termination is apparently uncommon. The younger the patient, the less definitely can this form in its chronic stage be diagnosed from congenital hydrocephalus. If the affection occurs during or after an acute infective illness, it may be difficult to diagnose it from so-called pseudo-meningitis or *méningisme* (see p. 760).

In other cases, by no means rare (Oppenheim,[1] Eichhorst, Boenninghaus, Bramwell, Brasch,[2] Schultze, Prince,[3] Münzer, Diller,[4] F. Krause-Böttiger, D. Gerhardt,[5] A. Fuchs,[6] Cramer,[7] Nonne, etc.), the disease *simulates a brain tumour*, and so complete is the resemblance of the symptoms that it is impossible to mention any certain point of difference. In these cases the diagnosis of brain tumour has practically always been given. Optic neuritis or choked disc and atrophy is an almost constant symptom. It was absent, however, in a case described by Bresler.[8] The affection of sight appears in many cases as a bitemporal hemianopsia. This is explained by the fact that the floor of the third ventricle, which bulges out like a cyst, specially compresses the middle portion of the optic chiasma. I have found it so reduced in one case that only two thin thread-like processes showed the course of the optic nerve. Simple blindness may also occur, and I have come to the conclusion from several cases, that it often develops acutely, or that the amblyopia may rapidly increase into amaurosis. In one of my cases it is said to have developed as the patient was bending down. Headache of great, but of varying intensity, vomiting, attacks of vertigo and spasms, paralysis of the cranial nerves (oculo-motor, olfactory, facial, and trigeminal nerves), exophthalmus, slow or accelerated pulse are the symptoms in the majority of the recorded cases. In only one of my cases was the headache slight. From the

[1] *Charité-Annalen*, xv., and *M. f. P.*, xviii.
[2] *Z. f. kl. M.*, Bd. xxxvi.
[3] *Journ. Nerv. and Ment. Dis.*, 1897.
[4] *Journ. Nerv. and Ment. Dis.*, 1898.
[5] *Therap. d. Gegenw.*, 1903.
[6] *Obersteiner*, xi.
[7] *M. f. P.*, xvii.
[8] *N. C.*, 1898.

published cases and one described by himself, A. Fuchs includes *tinnitus aurium* (and a corresponding objective blowing murmur sometimes occurs) among the symptoms of this disease. *General weakness in the extremities*, especially in the legs, is a very frequent and often an early symptom, and there may also be pain in the legs. In several of the cases under my observation there was a general rapid tremor, especially associated with active movements. Occasionally very intense pain, vertigo, and vomiting have occurred when the head has been bent backwards. In one of my cases there was also girdle pain, tenderness to pressure in the spinal column, etc.

There is nothing characteristic in any of these symptoms. They may also be produced, *e.g.* by a tumour of the cerebellum associated with hydrocephalus. There are only two factors which serve as guides to the differential diagnosis. One is that the hydrocephalus depends in many cases upon some congenital condition, which is shown by an *abnormal size and shape of the skull*. The second is the occurrence of *remissions and intermissions* of years' duration which are at least unusual in cerebral tumour. Thus I have described a case in which the severe brain symptoms gave place for a period of three years to a condition of comparative health, and only returned during pregnancy. Quincke and Weber have specially emphasised the variation in the intensity of the various symptoms. The absence of *focal symptoms* is also remarkable, if we except cerebellar ataxia, which is often mentioned (and the unilateral exaggeration of the knee-jerk noted by Grober [1]). Although focal symptoms may appear for a time they do not develop progressively, as in tumour. On the other hand, the *basal cranial nerves* are usually involved as the result of compression.[2]

Further observations are needed to show whether other symptoms, such as the early onset of the muscular weakness in the lower extremities, the tremor, and the exophthalmos, which is often present but usually slight, may be used as diagnostic signs. Examination of the cerebro-spinal fluid may also be helpful, as the amount of albumen which it contains is increased in tumour, and is normal in hydrocephalus, but this is not a factor upon which we can depend (see below).

Unilateral hydrocephalus, or hydrocephalus limited to one cerebral ventricle, occupies a special position. A few cases only of this kind have been reported by Mohr,[3] White, Spiller,[4] etc. During the last few years, however, Cramer and Weber have greatly extended our knowledge, as they have shown that many morbid processes in the region of the ventricular walls (tubercular, encephalitic, arteriosclerotic, gliomatous foci, etc.) may give rise to the development of unilateral hydrocephalus, limited to, or most marked in one ventricle. The symptoms are varied, but in general they have a great resemblance to those of brain tumour.

[1] *M. m. W.*, 1900.

[2] F. Krause has also observed a tremor limited to the right arm, but his case does not seem convincing. In a case of this kind I decided in favour of a brain tumour, as in addition to the general symptoms of cerebellar ataxia, there was also a certain tendency to conjugate deviation towards one side, and exaggeration of the tendon reflexes in the opposite leg. The autopsy, however, revealed an acquired hydrocephalus. When describing this and a similar case, I have carefully discussed the question (in the *M. f. P.*, xviii.), and have suggested that the labyrinth may also be involved in the process, and that when it is specially so on one side, unilateral pseudo-cerebellar symptoms (nystagmus, etc.) may occur. Circumscribed tenderness of the skull on percussion may also appear in chronic hydrocephalus (Oppenheim, Finkelnburg, *Z. f. N.*, Bd. xxix.).

[3] Casper's "Woch.," 1842; quoted after Uhthoff. [4] *Amer. Journ. Med. Sc.*, 1902.

The course, however, is usually very protracted and irregular, some of the focal symptoms are very inconstant, and intercurrent pyrexia occurs. Weber says: "If the course is slow, if the constant focal symptoms are accompanied by others of varying intensity and distribution, and if in addition there are definite general symptoms, the diagnosis of unilateral internal hydrocephalus is suggested."

There are cases of acquired hydrocephalus with acute exacerbations occurring after a chronic course. This acute relapse may, in cases with defective history, simulate an independent disease. Death may take place within some months or years. In one of my cases the disease lasted for nine years. Further, Quincke notes that there are very mild cases of this kind, in which the only complaint for years is one of headache, or possibly vertigo, and there is no doubt that in the absence of any objective symptoms, a diagnosis of neurasthenia is often given.

The difficulties of diagnosis are sufficiently apparent from what has been already said. They are not so great in childhood, when the size of the skull is increased. Rupture by the hydrocephalus of the closed sutures has been observed in a few cases of adults, but this is extremely rare. Fuchs has shown by one case the possibility of confusion with aneurism of the cerebral arteries. The diagnosis of *acute* serous meningitis is still less certain. Lumbar puncture seems to be a useful method for distinguishing it from other forms of meningitis, as in this case the fluid obtained is clear under high pressure (of 150 to 600 mm. of water and more), and it is usually rich in albumen and tends to coagulate, but contains few, if any, micro-organisms or cell elements. The data of different writers, however, vary greatly upon this point. Moreover, the diagnostic value of this examination is greatly lessened by the fact that an increase of the cerebro-spinal fluid and an increase of pressure within it has been observed in conditions of very different kinds (chlorosis, uræmia, sinus thrombosis, delirium tremens, etc.). Quincke thinks it probable that acute effusion into the ventricle may be a transient symptom in periodic headache (migraine), and may be somewhat analogous to acute circumscribed œdema of the skin. Kroenig, relying on Hansemann's investigations and his own observations, maintains that the lymphocytes are markedly increased in serous meningitis, and that this fact distinguishes serous meningitis from dropsy due to obstruction and from simple angio-neurotic accumulation of fluid. Quincke says that these effusions are usually of the same nature as normal fluid, but they may also resemble inflammatory exudations. Confirmation of Kroenig's statements would certainly be of great importance as regards diagnosis. Our conception of serous meningitis would thereby gain immensely in clearness, but it seems to me that, in spite of the very numerous recent investigations, this solution has not yet been reached.

We have in the previous section referred to the rare occurrence of *spontaneous escape* of the fluid through the nose. This is most apt to take place in the chronic form of acquired hydrocephalus and in tumour combined with hydrocephalus. Cases of this kind have been described by Paget, Baxter, Nettleship,[1] Smitt,[2] Nothnagel, Leber,[3] Wollenberg,[4] Caskey,[5] Freudenthal,[6] Hill, Halliburton.[7] Thomson, Coolidge,[8] Mignon,

[1] *Ophthal. Rev.*, 1883.
[2] *Ibid.*
[3] *Arch. f. Ophthal.*, 1883.
[4] *A. f. P.*, xxxi.
[5] *N. Y. Med. Journ.*, 1900.
[6] *D. A.*, Bd. clix.
[7] *Lancet*, 1899
[8] *Boston Med. and Surg. Journ.*, 1899.

De la Camp,[1] Schwab-Green,[2] and others. As a rule the fluid escapes from time to time in considerable quantities, or there may be a small but continuous dripping from the nose. It may drop constantly, though it is doubtful whether in such cases the base of the skull is always eroded, or whether the fluid escapes through preformed tracts (congenital dehiscence of the dura and the ethmoid bone, etc.). In a few cases there was no perforation of any kind, and it was suggested that the fluid had escaped by the lymph channels between the subarachnoid space of the brain and the perineural sheath of the olfactory nerve. It has been stated that this symptom may occur in individuals with normal brains (Coolidge). The troubles in the head, especially the headache, have often disappeared with the onset of the rhinorrhœa. In two of my cases in which this flow of fluid was very marked, the hydrocephalus was acquired; in a third, which was examined post-mortem, erosion of the ethmoid bone was distinctly evident, the meninges and parts of the brain obtruding through the opening.

Spontaneous recovery is very rarely brought about in this manner, but Glynn [3] has reported one such case.

The *prognosis* of acquired hydrocephalus is by no means absolutely hopeless. Cases of recovery are reported, especially in the acute form, and of improvement and arrest in the chronic forms. If we enlarge our conception as far as Quincke does, we should have to say that spontaneous recovery is very common. The danger to life increases in proportion as the clinical condition, through the progressive course of the disease, acquires a closer resemblance to that of brain tumour.

Treatment.—In the acute cases which resemble meningitis in their course or onset, the treatment is practically identical with that of meningitis. Quincke finds mercurial treatment very beneficial, and according to his descriptions it has been of great service, even in non-syphilitic cases. Concetti, Widal, Le Sourd, and Nonne agree with him. Heidenhain recommends the use of morphia on theoretic grounds. Münzer has seen good results from the application of hot packs to the head.

Lumbar puncture is very often recommended as a curative and palliative remedy (Quincke, Ziemssen, Lenhartz, Boenninghaus, Goldscheider-Peters, Mya, Kohts, Concetti, Immerwol, Chipault, Wertheimber,[4] Riebold,[5] Blumenthal,[6] Tobler,[7] Nonne, etc.). This is my own experience. In one very severe case of acute meningitis, Finkelnburg has seen recovery follow removal on two occasions of 30 c.cm. of fluid. I have also, in a case of chronic serous meningitis which simulated cerebellar tumour, seen permanent results from lumbar puncture, the headache, vomiting, oculomotor paralysis, and cerebellar ataxia disappearing completely, and the visual disorder, which was due to optic neuritis, being partially cured. The patient was able to attend to his business for five to six years (he died last year of some unknown cause).

Brasch, Henschen, Seiffer, Hirsch,[8] Gross,[9] Bokay,[10] and others have also obtained recovery by this method. It is specially indicated when the patient is threatened with blindness or the brain pressure reaches a height which endangers life. It may then be necessary to withdraw

[1] *B. k. W.*, 1904
[2] *Amer. Journ. med. Sc.*, 1905.
[3] *Brit. Med. Journ.*, 1905.
[4] *M. m. W.*, 1904.
[5] *D. m. W.*, 1906
[6] *A. f. Kind.*, Bd. xxxviii.
[7] "Korrespond. f. Schweiz. Ärzt.," 1904
[8] *W. kl. R.*, 1900.
[9] *A. f. Kind.*, xxvii.
[10] *Jahrb. f. Kind.*, 1903.

a large quantity at one operation (the pressure being carefully controlled), but otherwise the puncture should be repeated, a small quantity only being withdrawn each time. Quincke allows about 1 to 2 c.cm. to drop away in the minute, and when the initial pressure is high, he does not allow it to sink below 300 mm. Pilcz,[1] who has collected all the evidence up to 1899, and who is opposed to surgical treatment, has nevertheless been obliged to admit that lumbar puncture is justified in cases of this kind.

Ponfick (*B. k. W.*, 1905) has observed fatal meningeal hæmorrhage after lumbar puncture in hydrocephalus.

A few surgeons prefer direct puncture of the ventricles, especially when spinal puncture has given negative results and life is in danger. This method has come more into use during the last few years, thanks to Neisser's advocacy, and cases in which a severe condition was arrested in this way have been described by him, by Pollack, Hölker, and others. The symptoms have been checked in many cases by opening the dura mater of the brain. This result is most common in cases of serous meningitis associated with otitis media, as in a few which I have observed along with Jansen, but lumbar puncture has also sometimes been successful in such cases. Beck has seen the coma and Cheyne-Stokes respiration disappear immediately after puncture of the ventricle, and the power of sight return a few hours later. Grósz and Bókay have found that ventricular puncture has a palliative effect. F. Krause has trephined in hydrocephalus, which he took for cerebellar tumour, and marked improvement followed the operation.

Derivative methods of treatment are advisable in other cases. Thus I have repeatedly found a seton to be of service (see, however, p. 286), whilst Quincke gives the following advice as regards inunction with ointment of tartarated antimony :

An area of about the size of a florin is marked off on the cleanly shaven skull by a broad ring of adhesive plaster, and once or twice every day a bit of ointment the size of a pea is rubbed into this spot with a pad of gauze-wadding. Intense inflammation sets in, in two to four days, and leads to necrotic shedding of the tissue. The inunction should be stopped as soon as the signs of inflammatory swelling appear. After a few days œdema develops in the neighbourhood, and vomiting, albuminuria, and slight fever sometimes occur and rapidly disappear. Warm compresses aid the sloughing of the tissue, which is usually complete in ten to twelve days. Suppuration is kept up for six to eight weeks by ung. basil. Quincke lays special stress on the effect of this treatment upon the headache and the mental condition, but on the whole his method corresponds so little to modern ideas that his advice can hardly be said to have fallen on fruitful ground.

Syphilitic Diseases of the Brain

Literature : Heubner, " Die luet. Erkr. der Hirnarterien," etc., Leipzig, 1874 ; Wunderlich, " Die luet. Erkr. d. Gehirns und Rückenmarks," " Volkmanns Sammlung," etc., 1875 ; Fournier, " La syphilis du cerveau," Paris, 1879 ; Rumpf, " Die syphilitischen Erkr. d. Nervensyst.," Wiesbaden, 1887 ; Oppenheim, " Zur Kenntnis der syphilit. Erkr. d. zentr. Nerv.," Berlin, 1890 ; *Ibid.*, " Die syphilit. Erkr. des Gehirns," Nothnagel's " Handbuch," 2nd ed., Vienna, 1903 ; Nonne, " Syphilis und Nervensystem," Berlin, 1902. The monographs by Nonne and Oppenheim contain a comprehensive bibliography.

We do not here include affections which are connected with syphilis merely in their etiology, their anatomo-pathological basis consisting not

[1] Resumé *C. f. Grenzgeb.*, 1899.

in specifie changes, but in simple inflammation and degeneration. The following description applies only to true specific diseases of the brain.

Although Erb will not admit that this is a correct distinction, we still maintain that we are justified in adhering to it for the present.

There are many varieties of specific disease of the brain. In the majority of cases, syphilis of the skull bones being excepted, they arise from the *meninges* and the *vascular system*. We may have to deal with a diffuse, superficial, inflammatory new growth, or with circumscribed, solitary, or multiple tumours, or possibly with a combination of both these forms.

Syphiloma or *gummatous tumour* is a round, irregular, uneven, nodular growth, on the average about the size of a hazel or walnut, although it is occasionally larger. On section the periphery is greyish red, whilst caseation has taken place in the yellow, dry, tough focus in the central parts. The tumour is also often permeated with a compact fibrous tissue, which may extend into the vicinity in the form of an indurated membrane. The *vascular disease*, which is either an isolated affection or is associated with the tumour-formation just described and with meningitis, is an *arteritis*, which chiefly affects the basal cerebral arteries.

These processes, which have been studied in particular by Heubner, vary in their nature and genesis (see Figs. 356, 358). Gummatous tumour and meningitis may at first extend directly to the arterial wall. There is often in addition an independent disease of the arteries in the form of an end-arteritis, a proliferation arising from the cells of the intima, which gives rise to narrowing and even to obliteration of the lumen of the vessel, or is combined with thrombosis. The middle and outer coats may also be affected (mesarteritis, periarteritis). An arteritis or periarteritis gummosa (Baumgarten, Marchand) is also described, with the formation of nodes in the vessel wall, which are the products of a primary round cell accumulation in the outer coats, with caseation of the central parts. Further, all these different forms of vascular affection may co-exist. These processes are distinguished from the ordinary endarteritis deformans by the absence of fatty degeneration and calcification, but obliterating endarteritis is by no means always due to syphilis (Friedländer). A comprehensive review of this subject will be found in Nonne. Further, Hansemann, who thinks the affection commences in the perivascular lymph spaces, Benda (*B. k. W.*, 1904), Fabinyi (*Z. f. N.*, xxx.), Düring (*D. m. W.*, 1904), and Bruhns (*B. k. W.*, 1906) have studied the subject. *Phlebitic* processes of a similar kind have been found in the brain by Bartels, who has shown the probability of their connection with syphilis. Changes of this kind had previously been discovered in the veins of the spinal cord by Greiff (*A. f. P.*, xii.) and others.

The chief form of brain syphilis is *basal, gummatous meningitis*. It usually arises from the subarachnoid tissue in the *region of the chiasma*, from the space between the cerebral peduncles, and thence it extends more or less widely in a diffuse, although irregular manner, over the base of the brain. The new growth has partly a gelatinous, yellow, and partly a mottled appearance. At some points, and often over a wide extent, it forms a firm, connective tissue induration, which adheres firmly to the basal parts of the brain. It penetrates into all the bifurcations and depressions and spreads like a veil over the origin of the cranial nerves. Careful examination shows that these nerves, the *optic* and *oculo-motor* in particular, are not only surrounded by the tissue of the tumour but are themselves altered, swollen entirely or in parts, and show on section a vitreous-grey or mottled yellow colour. It may happen, however, that the cranial nerves enclosed within the tumour have a normal appearance to the naked eye. The large *arteries* at the base of the brain are often

involved in the process. Their walls are thickened and adhere to the diseased meninges, and their lumina are narrowed (or partially dilated). The diffuse meningitis may be associated with a circumscribed gumma, *e.g.* on a cranial nerve. The basal-meningitic process may also be limited to a small area, *e.g.* to the neighbourhood of the oculo-motor nerve, the chiasma, etc. On the other hand, a great mass of gummatous tumours may be found (Fig. 355) in a few cases, such as one described by Siemerling; the brain otherwise may be macroscopically intact, or it may show one or more *foci of softening*, especially in the region of the central ganglia or in the pons. *Hæmorrhages* or "*gummosities*" are also not uncommon. There may even be very extensive diffuse softening.

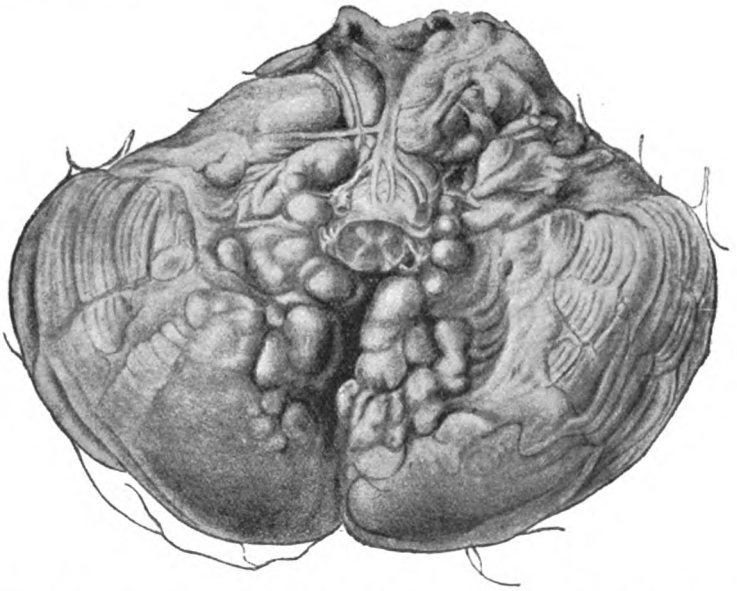

FIG. 355.—Syphilitic basilar meningitis and multiple gummata on the base of the brain. (Partly from figure of a case of Siemerling's.)

Apart from the diffuse and circumscribed forms, the distribution may be a disseminated one, taking the form of yellow or grey plaques (meningo-encephalitic foci) or of multiple tumours. Miliary gummata is a rare form of the syphilitic process; they may be scattered through the membranes of the brain and spinal cord. Mixed forms of a diffuse and disseminated process have also been described (Buchholz, Haenel, Barret, Babinski-Nageotte, etc.). In very rare cases suppurative meningitis has been thought to be connected with syphilis, but here the infection may have been a mixed one. It must be admitted that it may be a difficult matter to distinguish these syphilitic affections from certain forms of *tuberculosis* (Williams, Hoche, Leimbach, Schamschin, Böttiger) and of sarcomatosis (Schulz, Coupland-Pasteur, A. Westphal, Richter, Nonne, Redlich). The existence of bacilli, in addition to the condition of the other organs, would decide in favour of tuberculosis, just as the spirochæta pallida (*q.v.*) would indicate the syphilitic nature of the disease. It has recently been shown that serum reaction may be employed on the cadaver to prove the syphilitic origin of the disease. We cannot here describe the histological characteristics of diffuse sarcomatosis, but would refer the reader to the special memoirs on this subject, especially to that of Nonne. *Cysticercus-meningitis* (Askanazy, Rosenblath) resembles syphilitic meningitis in many ways, but is sufficiently distinguished by the presence of the parasite.

Histological examination shows the new growth to consist of an exuberant, vascularised *granulation tissue, rich in cells.* This tissue has in parts become caseous, and in others shows a fibrous transformation, but it is practically never found to have suppurated. Its relation to the vessels (Figs. 356, 357, 358) and to the nerve-roots (Figs. 359, 360, 361) is very typical. The round-cell proliferation continues into the adventitia of the vessels and to the *epineurium* of the nerves, especially the *optic* and *oculo-motor.* The thickened and infiltrated epineurium sends its processes from every side between the bundles of nerve fibres. These processes are septa of connective tissue, very much thickened, infiltrated

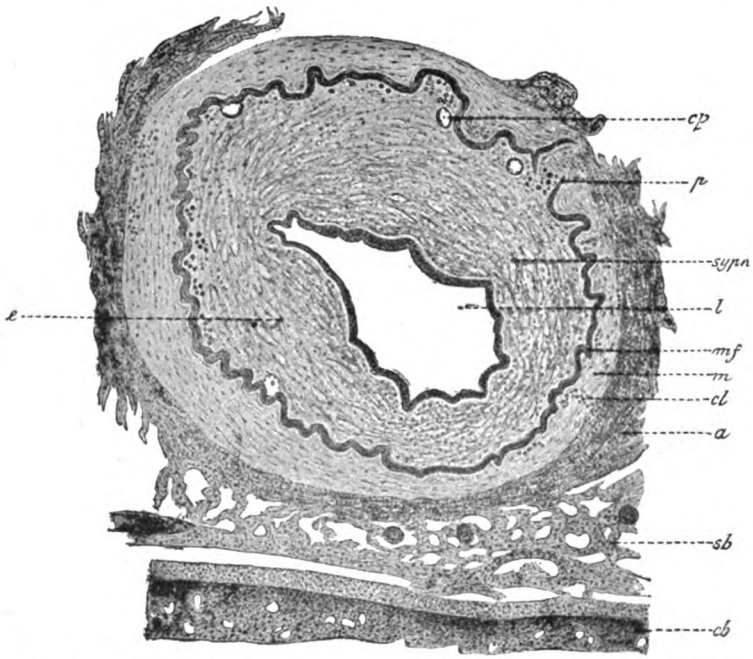

FIG. 356.—(After Heubner.) Syphilitic arteritis. Transverse section of a branch of the artery of the Sylvian fissure. *syph.* = syphilitic neoplasm ; *l*, lumen. The syphilitic neoplasm of the intima consists of cell-nets which are less thick at *a* ; *mf*, original fenestrated membrane ; *m*, muscular coat ; *a*, infiltrated adventitia ; *sb*, infiltrated subarachnoid space ; *cb*, surface of the brain ; *cl*, round cells ; *cp*, newly formed capillary ; *p*, pigment. (Carmin stain.)

with small cells and rich in vessels. Whilst some of the nerve fibres become atrophied from the pressure of the tumour and its processes, the nerve is swollen, possibly to four or five times its normal size, from infiltration by the newly formed tissue, but during the later stages it may become atrophied. The swelling may be due partly to œdema. It is obvious that the superficial layers of the brain itself may be directly involved by the disease of the meninges. Gummata from the membranes may penetrate deeply into the brain substance.

The same process of a gummatous meningitis, sometimes circumscribed, sometimes spreading over a considerable area, may develop, on the *convexity* of the brain, though not so often as at its base. In this case it penetrates more or less deeply into the tissue of the brain and thus

affects its functions. *Syphilitic meningo-encephalitis* may lead to softening of a whole lobe of the brain, and indeed of a whole hemisphere. It has also been observed that the same process may involve the base and the convexity of the brain simultaneously A *multiplicity of lesions is*, to a certain extent, characteristic of cerebral syphilis.

Gummatous tumours seldom have their origin in the interior of the brain. But it is very remarkable that *gummatous neuritis of the cranial nerves*, especially the optic and oculo-motor, may be a primary, independent disease. *Syphilitic arteritis* of one or more of the cerebral arteries is not rare, and it may not be associated with any appreciable changes in the brain and its appendices. This arteritis may indeed be limited to small branches of an artery, *e.g.* of the basilar artery (Oppenheim-Hoppe, Henneberg), and this circumscribed affection may be the only sign of the brain syphilis (compare Fig. 358). Runeberg thinks that localised arteriosclerosis is usually of specific origin.

In these local circumscribed affections, we should always bear in mind that syphilis has the tendency to produce diffuse and disseminated changes in the brain, especially in the meninges,

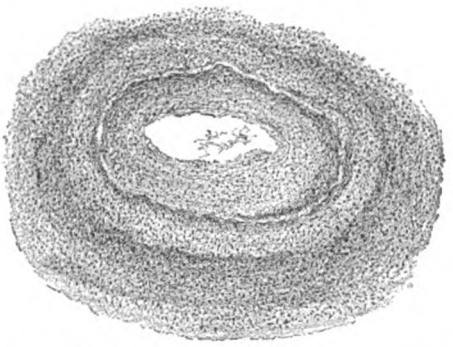

FIG. 357.—Arteritis from syphilitic basilar meningitis. Transverse section of an artery. (Stained with carmin and alum-hæmatoxylin.)

which can only be detected by microscopical examination. Babinski and Nageotte (*Nouv. Icon.*, xvi.) have again drawn attention to this fact.

As regards the histological details, and especially the histological differentiation of cerebral syphilis from general paralysis, we would specially refer to the work of Nissl and Alzheimer (see following chapter).

Circumscribed foci of softening which cannot be attributed to a vascular disease and are possibly of encephalitic origin are found in exceptional cases.

Etiology.—Syphilitic diseases of the brain have their origin in *constitutional syphilis*.

It is very doubtful whether they may arise from a soft sore, although Hitzig has made the following suggestion : " In venereal infection, several poisons are from the first inoculated, or are produced in the first stage of the infection. A toxin, which is present at the commencement of the primary sclerosis or its derivatives, gives rise to the secondary and tertiary late forms of syphilis. Another poison which may be, although it is not necessarily, present in the same syphilitic ulcer or its derivatives, is the cause of a peculiar morbid change in the composition of the blood, which, after an interval of years and decades, predisposes the whole nervous system to degenerative

changes. The same toxin may not only be contained in the primary syphilitic lesion, but also in the cancerous ulcer. This theory, however, does not apply to cerebral syphilis in the strict sense of the word.

Our knowledge of the nature of syphilis has greatly advanced during the last few years. The possibility of experimental study of this disease and of the toxins and anti-toxins which it generates in the body is due to the fact that Metschnikoff and Roux,[1] Neisser,[2] Lassar,[3] and others have succeeded in transmitting syphilis to apes. This was followed by the discovery by Schaudinn and Hoffmann,[4] of the spirochæta pallida, which has become all the more important as an evidence of the syphilitic nature of an organic disease since it has been found to be present also in congenital syphilis (Levaditi, Buschke, Ranke, Babes [5]). We must also remember the results of lumbar puncture, or examination of the cerebro-spinal fluid (see p. 161, also p. 761 *et seq.*). The investigations of Wassermann-

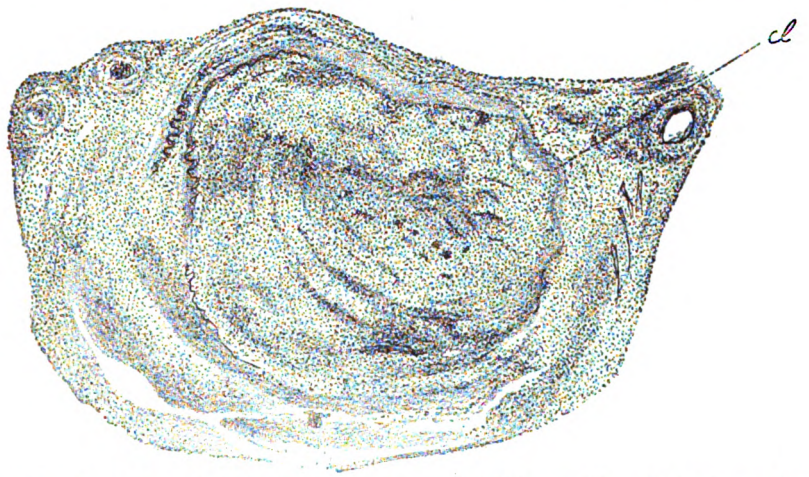

FIG. 358.—Thrombosis of the basilar artery due to syphilitic arteritis. *el*, elastic membrane.

Neisser-Bruck,[6] which are supplemented by those of Marie-Levaditi, Plaut,[7] Citron,[8] etc., have greatly deepened our knowledge and contributed to the advancement of the diagnosis, as they have succeeded in identifying a serum-reaction which occurs almost exclusively in syphilis. Naturally this does not by any means dispose of all the difficulties. The presence of the spirochæta cannot always be demonstrated in all the products and stages of syphilis, and moreover, the specific reaction of the fluid, *blood*, and tissue juice of syphilitics is not a constant symptom, so that no imperative, decisive significance is attached to a negative result of this test, even by those who are responsible for its discovery. Finally, some of these reactions—the cytological and chemical findings in the fluid (Nonne-

[1] *Ann. de l'Institut Pasteur*, 1903 and 1904. [2] *D. m. W.*, 1904 and 1906.
[3] *B. k. W.*, 1903 and 1904. See also E. Hoffmann, *B. k. W.*, 1905.
[4] *D. m. W.*, 1905, and *B. k. W.*, 1905. We cannot here enter into their controversy with Siegel and his theory of "Cytorrhyktes luis."
[5] *B. k. W.*, 1905.
[6] *D. m. W.*, 1906; see also Wassermann-Meyer, *D. m. W.*, 1907, which contains an exact description of the nature and the method; Blaschko, *B. k. W.*, 1908.
[7] *D. m. W.*, 1907, and *M. f. P.*, xvii. [8] *B. k. W.*, 1907.

Apelt[1]), and Wassermann's serum-reaction, occur with as great, if not greater regularity in the so-called metasyphilitic diseases.

The experience of many writers (Lavallé, Brosius,[2] Nonne, Erb, etc.) seems to suggest that a certain form of syphilis is specially apt to attack the nervous system.

The various syphilitic affections of the brain usually appear within the first few years after the infection. A large percentage occur within the first or the first two years. The nervous system is seldom affected after the tenth year (Naunyn). Brain symptoms have appeared in a few cases at the commencement of the secondary stage, a few months or even a few weeks after the infection (Nonne, Saenger, Gilles de la Tourette, Mingazzini, Oppenheim, E. Hoffmann, Finkelnburg, etc.).

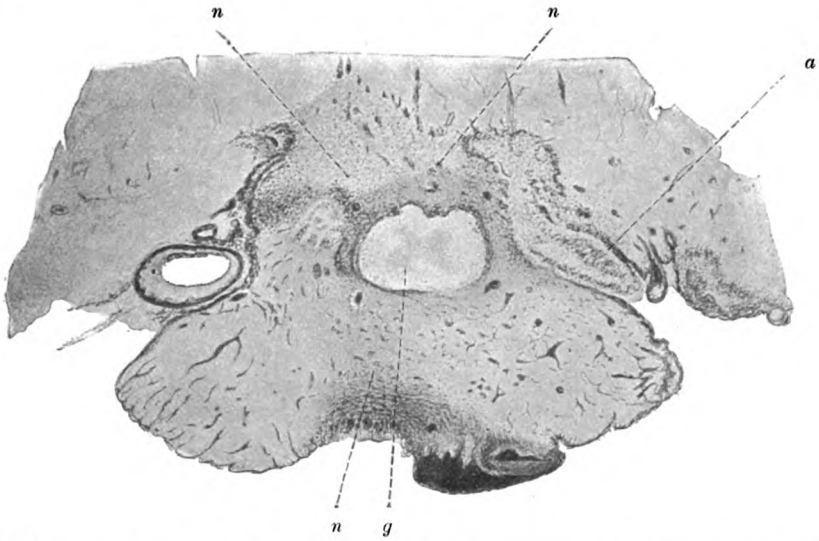

FIG. 359.—Syphilitic neoplasm above the optic chiasma, extending specially over the middle portion. *n*, neoplasm; *a*, thrombosed artery; *g*, gumma.

Henschen states that of 754 syphilitics treated in the hospital of Helsingfors, 112 suffered from syphilis of the brain.

Trauma (injury to the head), mental exhaustion, emotion and alcoholism make the brain more liable to suffer from syphilis. It is especially noted that the first signs of cerebral syphilis often appear directly after an injury to the head.

Symptoms.—We shall first describe the typical form—gummatous basal meningitis—and then the others. The *clinical condition* produced by this diffuse basal syphilis of the brain is a very characteristic one, in spite of the great variety of the symptoms. Of the *general symptoms* headache is the most important, and is the most constant and early sign. It becomes at times exceedingly severe. The exacerbations often occur at night. Vomiting and vertigo may follow, and there are often attacks of *loss of consciousness* and general convulsions. As a rule the mind is affected, a moderate degree of dementia, weakness of memory,

[1] *A. f. P.*, Bd. xliii.

[2] *D. m. W.*, 1903.

and apathy being evident. On the other hand we do not observe the persistent, gradually increasing stupor which is characteristic of most other forms of intracerebral tumour. The patient is quite conscious during long intervals of the course, but he suffers from intercurrent attacks of impaired consciousness, *e.g.* deep stupor, lasting for several hours or days, or conditions which cannot on superficial observation be distinguished from sleep, dreaming or intoxication, or *attacks of violent excitement*, confusion, and delirium. An alternation of delirium with comatose conditions is specially characteristic, and it should be noted

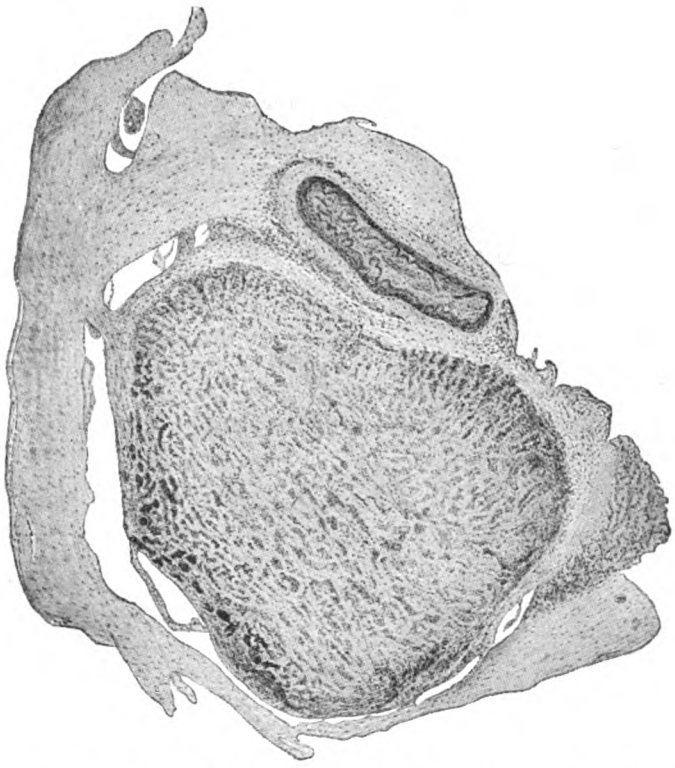

FIG. 360.—Transverse section of the optic nerve near the bony canal. Marked perineuritis, neuritic atrophy, obliteration of the ophthalmic artery. (After Uhthoff.)

that even deep coma may disappear and the patient become fully conscious.

Symptoms of paralysis, which point to an affection of certain cranial nerves, also appear. They may develop along with the general cerebral symptoms, but they usually follow, and only in rare cases precede them. The fact that the *optic nerve* and the *nerves of the ocular muscles*, especially the oculo-motor, are by far the most frequently affected, and often exclusively so, is in accordance with the anatomical conditions. There is paralysis or paresis of the whole oculo-motor nerve or some of its branches. The abducens and trochlear nerves are much less often affected, the latter usually on one side only. The paralysis in many cases attacks all the

branches of the oculo-motor nerve simultaneously, but in others we can observe one ocular muscle becoming affected after another. Ptosis is specially common. The pupillary nerves may be alone involved, or loss of the pupil reflex may be the only remaining symptom of the brain syphilis, as in a case under my observation in which for fifteen years it was the only sign of the previous cerebral syphilis. But on the whole pure internal (and external) opthalmoplegia is a rare symptom in basal syphilis, although some authors think that this is its main cause (Bumke).

If both oculo-motor nerves be involved, one side is almost always affected more than the other, and some of the other cranial nerves are usually involved on the same side.

Disease of the optic nerve gives rise to *ophthalmoscopic changes and functional disorders*. Uhthoff [1] found this nerve affected in 14 out of 17 cases of brain syphilis which were examined post mortem, and he observed pathological ophthalmoscopic changes in 40 per cent. of the cases which were only examined clinically. Unilateral or bilateral optic neuritis, typical choked disc and atrophy of neuritic origin are very common. Simple (descending) atrophy has also been found in many cases, but Uhthoff thinks it improbable that a pure primary progressive atrophy of the optic nerve may occur in cerebral syphilis. Ophthalmoscopic examination often yields a negative or indefinite result, whilst testing the sight shows marked disorders. Thus, according as the tract or the chiasma are affected, we find homonymous hemianopsia of one eye with temporal hemianopsia of the other, and finally there may be blindness on both sides. This is rarely total and persistent, but a temporary amaurosis is not an unusual symptom. A great number of such cases have been described by Oppenheim, Uhthoff, Siemerling, Knotz, etc. If the disease arises from the optic nerve itself, it may cause concentric or irregular narrowing of the field of vision, diminution of the central acuity of vision, central scotoma (Uhthoff, Wilbrand), etc.

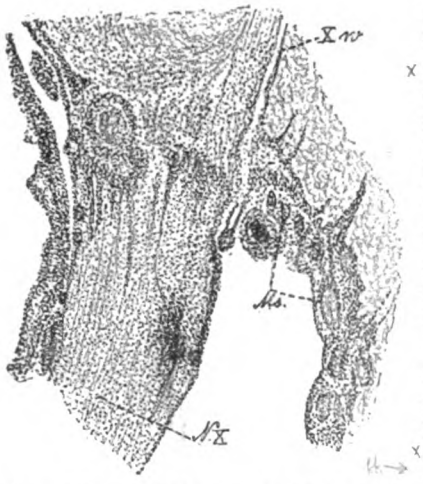

FIG. 361.—Syphilitic meningitis extending to the medulla oblongata and vagus root. (Stained with carmin and alum-hæmatoxylin.)

The *olfactory nerve* is often embedded in the new growth, the result being unilateral or bilateral *anosmia*. Affection of the fifth nerve, especially on one side, is still more common, and this gives rise chiefly to the irritative symptoms of neuralgic pain and hyperæsthesia, but hypæsthesia and anæsthesia are not unusual, and *neuroparalytic keratitis* has been observed in a good many cases.

Should the process extend still further back, the *facial* and *auditory* nerves are involved. The facial paralysis is naturally of a peripheral

[1] "Über die bei der Syphilis des Zentralnervensystems vorkommenden Augenstörungen," Leipzig, 1894. See also the works of Wilbrand-Saenger and of Terrien, "Die Syphilis d. Auges und seiner Adnexe." German edition, München, 1906.

character, although changes of the electrical excitability are not always present. Facial diplegia may also develop from cerebral syphilis. Should the meningitis extend chiefly into the posterior cranial fossa, the corresponding nerves from the medulla oblongata (*hypoglossal, vagus-accessorius*, etc., will be involved, see Fig. 361), and symptoms of their paralysis will develop.

The *polydipsia* and *polyuria*, which are common symptoms of basal syphilis, do not necessarily indicate an affection of the medulla oblongata and the vagus. They are often observed in syphilitic processes which involve the region of the chiasma, or the floor of the third ventricle.

We shall not discuss the occurrence of transitory glycosuria and diabetes mellitus in syphilis.

It still remains to be said, however, that the symptoms do not by any means always indicate that the process of new growth is one of gradual extension in the sense of involving only the cranial nerves which have their origin and course in the same neighbourhood. On the contrary, the symptoms often point to separated foci. This is partly explained by the fact that the nerves enclosed in the new growth are by no means equally injured in their structure and function.

These symptoms correspond to a morbid process in the base of the brain, which causes compression and spreads over a large area, but they do not thereby reveal the specific nature of the process. Naturally, other tumours very seldom extend in the same manner. Tubercular meningitis is excluded by the development, course, and condition of the temperature, as in syphilitic meningitis the *temperature* as a rule is *normal*, or merely slightly raised in exceptional cases. Some few cases have certainly been published (Richter, Sidney, Caro), which make it probable that fever is one of the rare symptoms of constitutional syphilis, and may for a long time be its only manifestation.

None of these factors, however, are so decisive as the peculiar course of brain syphilis, the *inconstancy of the symptoms, and the way in which they come and go and shift about.* I [1] have found this *variation* to be very marked in regard to the visual disturbances. Thus in a few cases, examination on different days showed sometimes a normal field of vision, sometimes an irregular concentric narrowing, with margins which differed from day to day, sometimes a marked hemianopsia, etc.

Transient bitemporal hemianopsia seems to me to be peculiarly characteristic. Temporary and recurrent amaurosis may occur, as well as recurrent choked disc. The oculo-motor paralysis is also very variable. It may be present one day, and may greatly improve a few days later, only to return after a short interval in its full intensity and completeness. I have treated a patient in whom ptosis and paralysis of the superior rectus of one eye repeatedly appeared, and again disappeared after a duration of ten minutes to half an hour, under my immediate observation. In a few cases the *loss of the pupil reflex* has been found to come and go (Buttersack, Oppenheim, Nonne). In one case I have seen repeated sudden onset and disappearance of a superior alternate hemiplegia along with persistent partial oculo-motor paresis. The facial paralysis may relapse several times and may even, as I have sometimes seen, pass from one side to the other.

[1] *B. k. W.*, 1887 and 1888.

This rapid variation of the symptoms is explained by the pathological changes. The granulation tissue develops, proliferates, and dies in quick succession, and this process is constantly being repeated. The nerve which is caught in this tissue is therefore subject to pressure which is more variable than in any other disease.

The symptoms described so far are directly produced by the meningitis and the gummatous neuritis or perineuritis of the cranial nerves. The symptoms which are due to disease of the *vascular system*, and which have been specially studied by Heubner and Rumpf, are of great importance. The most constant of these is *hemiplegia*, which may appear at any time, but develops as a rule only in the advanced stages. In the nature of the process, the hemiplegia is usually preceded by other premonitory symptoms. Circulatory disturbances first appear, followed by definite occlusion of the vessel and consequent softening of the area thus deprived of nutrition. The hemiplegia is, therefore, preceded by slight *apoplectiform attacks*, paræsthesiæ, and slight paralysis in the corresponding half of the body, and finally the hemiplegia, which at first is merely *transient*, becomes established after a fresh attack. The onset of the hemiplegia in one limb after another is also typical. Within a day or two the leg, then the arm, and then the facial nerve, etc., become successively paralysed. The mind may be unaffected, or there may be impairment or complete loss of consciousness. As the arteries are chiefly affected on the side on which the basal process is most advanced, the hemiplegia usually appears on the side *opposite* that of the paralysis of the cranial nerves, and it should be carefully noted that the principles laid down for the localisation of so-called alternate (superior and inferior) hemiplegia should be applied with great caution to cases of cerebral syphilis. Thus, paralysis of the oculo-motor of one side and of extremities of the other may in this case sometimes be caused, not by a focus in the cerebral peduncle, but by a specific basal lesion and the vascular conditions to which it gives rise. The hemiplegia may, however, involve the side of the body corresponding to that of the paralysis of the cranial nerves.

In not a few cases *hemianæsthesia*, *aphasia*, and *hemianopsia* of cerebral origin are due to the same cause, *i.e.* to the endarteritis. As this endarteritis is severe and often widespread, it is not surprising that the extremities should sometimes be paralysed on both sides. Nor, when the *basilar* and *vertebral arteries* are chiefly involved, is it uncommon for the pons and oblongata to be affected by the secondary vascular condition, so that bulbar symptoms appear in addition to those already described. These areas may also be directly involved by the gummatous process.

This is the *typical picture of syphilitic basal meningitis*, but it may vary in many ways according to the distribution of the process, and to the cranial nerve or arterial branch that is chiefly affected.

But there are many cases in which the process is limited to a small area, *oculo-motor* paralysis or an affection of the optic nerve being the only objective signs in addition to the *headache*.

As regards the course of the disease, its development may be acute, subacute, or chronic. As a rule it is insidious, the headache preceding the onset of the paralytic symptoms for weeks, months, and even for years. The later course is very seldom acute; it is much more often subacute or chronic, and characterised by *repeated remissions* and *exacerbations*, the

disease thus consisting of a number of periods of severe illness, with intervals of complete or comparative good health.

The *prognosis* is not unfavourable. Recovery can, however, only be expected so long as *specific* changes in the tissue are the only ones present, the secondary conditions, viz. the atrophy of the cranial nerves produced by the compression, and the softening due to occlusion of the arteries, not having developed. If the general cerebral symptoms are only accompanied by signs of gummatous neuritis, *complete recovery* may take place. It may often be shown to have done so by the condition of the eyes, although some defect may be left behind. Thus optic neuritis or choked disc may completely pass away, whilst marked atrophy may remain. One can imagine that a cranial nerve which has been in a condition of paralysis for years, loses its power of complete recovery.

The prognosis as to recovery becomes much less hopeful when hemiplegia sets in. It may, indeed, completely disappear in its early stage, but the longer it persists the more improbable does its disappearance become. There is hardly any prospect of recovery after contracture has set in.

The *danger to life* is always *great*, and it increases in proportion as the process extends, either directly or through involvement of the basilar and vertebral arteries, into the vicinity and site of the pons and medulla. Even when the paralytic symptoms have entirely passed away, a certain mental weakness often remains. *Old age* and a *poor state of nutrition* have also an adverse effect upon the prognosis.

Syphilitic meningitis of the *convexity* only gives rise to characteristic symptoms when it extends over those parts of the cortex, lesion of which produces functional symptoms. One symptom may of course have its origin at any site, viz., the *violent*, persistent, and often *localised headache*, which sometimes corresponds to a localised tenderness of the skull to percussion. Otherwise it is specially apt to cause brain symptoms when it is situated in the region of the *motor* zone or of the *speech centre*, and these are, as a matter of fact, its favourite sites. If it occupies the region of the motor centres, it gives rise to symptoms of cortical epilepsy, associated at a later stage with monoplegia. Here, however, we have but few definite indications of the specific nature of the disease. The *relapsing course*, is, indeed to be noted. But it has a less decisive effect upon the diagnosis from the fact that new growths of the motor zone are apt to cause symptoms of a convulsive character: this is indeed in the nature of *Jacksonian* epilepsy. On the other hand, the variability of the symptoms is even more marked than in other diseases of the motor zone. The remissions are usually more complete and of longer duration, and they may occur during the paralytic stage. The second factor which is of diagnostic importance is the possible absence in specific diseases, even to the end of the disease, of general symptoms of intracranial tension, especially of choked disc. This is undoubtedly due to the tendency of the new growth to extend in a superficial manner. This factor also has a restricted diagnostic value, as (1) glioma of the cortex may be in existence for a considerable time before any change appears in the fundus of the eye, and (2) a syphilitic cortical process is often associated with a corresponding basal process (Oppenheim, Schaffer,[1] Babinski), which

[1] *N. C.*, 1904.

may directly involve the optic nerves. Thus Nonne and I have found choked disc in several cases of syphilitic cortical epilepsy. It lies in the nature of things that irritative symptoms, epileptiform attacks, persistent contractions in certain muscle groups, tremors, contracture, pain in the opposite side of the body, etc., should be very prominent features.

The fact that the *distribution* of the disease is manifested by symptoms extending over a large *superficial area* is often of assistance in the diagnosis.

I have observed cases in which unilateral headache first appeared, followed after a time by convulsions in the opposite side of the body, then by transient monoplegia or hemiplegia, and finally by paralysis which was persistent but varied in intensity. During a fresh attack aphasia appeared, which at first was periodic, but finally became persistent. The intellectual impairment, which was considerable, also pointed to a widespread lesion.

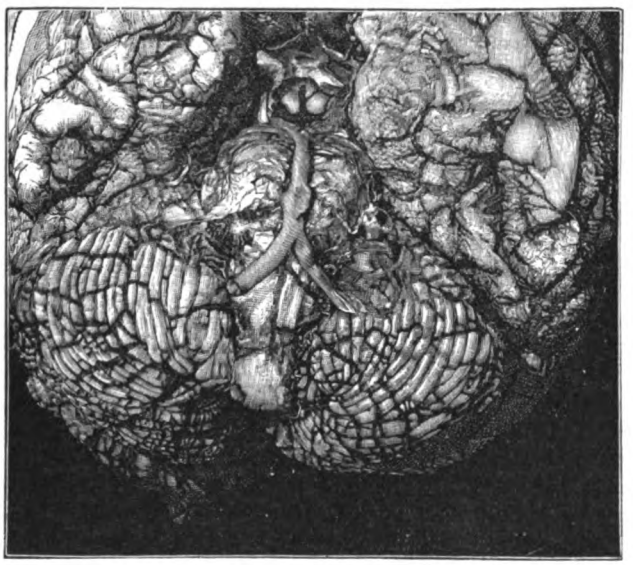

FIG. 362.—Localised arteritis of the basilar artery, with aneurysmal dilatation and thrombosis, the only evidence of cerebral syphilis. (After Henneberg.)

Such a development and grouping of symptoms always justifies the assumption of a specific process, and in such cases the results of treatment afford valuable confirmation of the diagnosis of cerebral syphilis. According to Widal, Sicard, Ravaut,[1] Babinski, Bèlétre, Milian, Merzbacher,[2] Ninon,[3] etc., this diagnosis will be still more definitely confirmed if the cerebro-spinal fluid shows an increase in the number of lymphocytes and in the amount of albumen (Nonne-Apelt). This seems also to be the case in hereditary syphilis (Tobler[4]). Even in simple headache, the syphilitic origin may be ascertained in this way (Widal, Milian-Crouzon, Marie-Guillain). Of course the same conditions may be found in metasyphilitic diseases, and so, even in a higher degree, may the Wassermann fluid or serum reaction.

[1] *Ann. de Dermat.*, 1903 and 1904.

[2] *C. f. N.*, 1905 and 1906. See the references on p. 766 and in the "Sammelreferat" of Raubitschek, *C. f. Gr.*, 1906. Consult also Erb, *Z. f. N.*, xxxiii.

[3] *Thèse de Lyon*, 1906.

[4] *Jahrb. f. Kind.*, xv. See also Ravaut, *Ann. de Dermat.*, 1907, and *Gaz. des hôp.*, 1907.

In a case described by Wernicke and Friedländer, a bilateral syphiloma in the temporal lobes gave rise to deafness. Should the disease develop in the occipital lobe, hemianopsia, etc., may be produced.

In extensive syphilitic meningitis of the convexity, the mental symptoms are a marked feature of the disease. The condition may indeed greatly resemble, or even be confused with paralytic dementia. In cerebral syphilis complete loss of the power of perception is often observed, the intelligence being otherwise unimpaired.

A condition corresponding to Korsakoff's psychosis has in one case been attributed by Roemheld (*A. f. P.*, Bd. xli.) to cerebral syphilis. As to the mental disturbances in brain syphilis, consult Nonne, Ziehen (*B. k. W.*, 1905), and the following chapter.

The *prognosis* is on the whole more favourable in cases which show this localisation (apart from the diffuse processes mentioned above) than in basal syphilis. I have seen many such cases recover under treatment. In some of these the headache was only accompanied by aphasia, which developed in successive stages; in others there were symptoms of cortical epilepsy and monoparesis (with or without disorders of sensibility), or of aphasia and facio-brachial monoparesis. In some of these cases the paralytic symptoms were cured, whilst the convulsive attacks persisted, and *vice versâ*.

Single gummatous tumours are seldom found in the brain; these tumours are generally multiple. The symptoms do not differ essentially from those of other kinds of new growths, apart from their periodic character, which is very marked.

The symptoms produced by primary syphilitic neuritis need not be specially discussed.

Kahler describes the symptoms of "multiple syphilitic root-neuritis" as follows: In addition to other symptoms of cerebral syphilis, or even without these, a gradually progressive paralysis appears in various cranial nerves, which may be recognised as *peripheral* (*e.g.* the facial). The paralysis involves one cranial nerve after the other in a quite irregular manner. Then follows as the result of lesion of the posterior roots, neuralgia in the region of various spinal nerves, which slowly increases in intensity, and is associated with cutaneous hyperæsthesia, girdle sensation, etc. Lesion of the anterior roots gives rise to corresponding motor paralyses of peripheral character, etc. This description is somewhat too precise.

Finally, there are not a few cases in which the *arterial system* is chiefly or solely affected. Symptoms often occur in such cases only when occlusion (less often rupture) of a vessel takes place. According as the thrombosis affects this or that branch, the symptoms take the form of *hemiplegia, hemianæsthesia, aphasia, hemianopsia*, or a combination of these, or *an acute bulbar* paralysis may develop after an apoplectic fit. The symptoms thus correspond in general to those of *cerebral softening*. There is usually, however, some indication of the specific nature of the disease. There are often *prodromata*, especially in the form of intractable headache and vertigo, and *apoplectiform attacks*, which leave behind them transient paralyses in the muscles which are subsequently definitely paralysed. These apoplectic attacks are sometimes followed by a condition of prolonged *stupor* and *confusion*. Further, the hemiplegia is sometimes associated with *dementia*, which could not be explained by a

mere circumscribed focal disease. *Immobility of the pupils* may also occur, and its presence is strongly suggestive of the syphilitic nature of the disease.

Of all the specific diseases of the brain, those involving the *vessels* give the gravest prognosis, but even vascular affections have been cured by anti-syphilitic treatment (Leudet). The symptoms caused by aneurisms of the basal arteries and their rupture have already been discussed. Nothnagel was able in one case to make a correct diagnosis from the blood in the fluid obtained from a syphilitic by lumbar puncture (see p. 935).

This tendency to the formation of aneurisms has been specially marked in a few cases, such as that of Panichi (*Riv. sper.*, xxviii.).

We should also point out that symptoms of syphilitic meningomyelitis may be associated with those of cerebral syphilis, especially with those of basal meningitis. Indeed this *syphilitic cerebro-spinal meningitis* is in our experience one of the most common forms of syphilitic disease of the central nervous system. The clinical features hardly require special description, as they can easily be inferred. The brain symptoms are occasionally so prominent that the spinal ones are entirely masked, or absence of the knee-jerk on one or both sides, unusual weakness in the legs, a girdle sensation, etc., are the only signs of a spinal process. In other instances the spinal symptoms are clearly developed, and the group of spinal symptoms described on pp. 307 *et seq.* may be combined with those described as cerebral. There is a large number of cases in which the disease of the spinal cord corresponds to the type of Erb's syphilitic spinal paralysis, and this in association with the cerebral symptoms gives rise to a clinical condition allied to that of disseminated sclerosis. The spinal disease may likewise correspond to the type of the Brown-Séquard unilateral lesion. In another group of cases the spinal symptoms resemble those of tabes dorsalis, and form along with the brain symptoms a special type, to which I have given the name of pseudotabes syphilitica.

The signs of *hereditary syphilitic* diseases of the brain usually appear during the first period of life, but they frequently do not become evident until the time of puberty. The brain disease may even develop in the third or fourth decades, as I have found in two cases. The pathological changes are practically identical with those described under acquired syphilis. All the symptoms there discussed occur here also, the disease being in this case characterised only by its severity and extent and by the inhibitions of development and secondary changes which it produces (atrophy, sclerosis, hydrocephalus, hypoplasia of certain parts, etc.). The symptoms are, therefore, practically identical with those of cerebral and cerebro-spinal disease due to acquired syphilis, except that naturally in this case inhibition of the mental development is a very outstanding feature. I have, it is true, seen conditions of this kind in which the intelligence was in no way affected. Epilepsy and its varieties is a very prominent symptom. The most important cases have been reported by Fournier, Rumpf, Hutchinson, Money, Bury, Jürgens,[1] Dowse,[2] Siemerling,[3] Boettiger, Zappert, Gilles de la Tourette, Gasne, Ashby, Heubner, Richon,[4] Cabannes, Tugendreich,[5] Ingelrans,[6] J. J. Meyer,[7]

[1] *Charité-Annalen*, x.
[2] "Syphilis of the Brain," 1881.
[3] *A. f. P.*, xx.
[4] *Nouv. Icon.*, xvi.
[5] *Jahrb. f. Kind.*, viii.
[6] *Gaz. des hôp.*, 1904.
[7] *Thèse de Paris*, 1904.

Raymond,[1] Quilliet,[2] Bresler,[3] Ranke,[4] etc. Opthalmoplegia interna and loss of the pupil reflex may be the only symptoms of the involvement of the nervous system in hereditary syphilis. When these are present in idiocy and infantile imbecility they always point, according to König, to a syphilitic basis. Family diseases also, in which the most prominent symptoms are dementia and spasticity, should be attributed to hereditary syphilis (Homén, Vizioli, etc.). The fact that hydrocephalus may develop from hereditary syphilis has been mentioned in the preceding chapter. I have had a case of hereditary syphilis under my care in which a condition corresponding to syringobulbia had developed, and which improved to a great extent under continued doses of iodide. Some forms of infantilism (*q. v.*) may also be traced to hereditary syphilis. With regard to the relations of juvenile paralysis to syphilis, the following chapter should be consulted.

The prognosis of hereditary syphilitic diseases of the brain does not essentially differ from that of cerebral syphilis in adults. It is true that syphilitic and metasyphilitic processes seem to occur together more frequently in the former case. Moreover, there is greater danger of an arrest of mental development. Nevertheless I have had an opportunity of observing many cases in which the symptoms of a severe brain disease of this kind have been completely cured by energetic antisyphilitic treatment.

As regards the *differential diagnosis* of cerebral syphilis, the chapters on neurasthenia, hysteria, multiple sclerosis, etc., and the following chapter should be consulted.

Catola (*Nouv. Icon.*, 1906) has recently maintained that there is a syphilitic form of disseminated sclerosis. See also Perrin-Parisot (*Rev. de Méd.*, 1907).

We must also point out that multiple and diffuse *sarcomatosis* may, especially according to Nonne's cases, produce a clinical picture closely allied in its symptoms and course to cerebral syphilis. The diagnosis in such cases must be made from the history of the case, the general physical examination, and the result of treatment. Lumbar puncture or serum reaction may possibly also afford valuable evidence. We should remember that this form of sarcomatosis is a rare disease as compared with syphilis.

According to Askanazy, Rosenblath, and Henneberg, basal cysticercus-meningitis has a still greater resemblance to syphilitic basal meningitis. The factors above mentioned are the essential points for diagnosis.

It may be very difficult to establish a differential diagnosis between cerebral and cerebro-spinal syphilis and tabes dorsalis, especially as regards the symptom of oculo-motor paralysis. If loss of the pupil reflex is the only symptom, and if it cannot be ascertained that it represents the residue of a past brain disease, there is every probability that the case is not one of a true specific process, but of commencing tabes (or paralytic dementia). Nevertheless this symptom may be the only change produced by the syphilitic virus in the nervous system (Chvostek, Erb, Oppenheim, Moeli, Nonne). It has also been described as the only sign of hereditary syphilis (Uhthoff, Nonne, Finkelnburg,[5] Oppenheim). I do not go so far, however, as Babinski and others, in regarding it

[1] *R. n.*, 1904.
[2] *Thèse de Bordeaux*, 1903-1904.
[3] "Erbsyphilis und Nervensystem," Leipzig, 1904. See also as regards the ophthalmoscopic changes in hereditary syphilis, Hirschberg, *D. m. W.*, 1906; Fairbanks, *Journ. Amer. Med. Assoc.*, 1907, and as regards the late form of Erb's syphilis, Fournier, *R. n.*, 1907.
[4] *A. f. P.*, Bd. xlii.; *N. C.*, 1907.
[5] *Z. f. N.*, xxiii.

in a general way as a proof of syphilitic infection. I have, for instance, seen it in a few cases of brain tumour in which there could be no suspicion of syphilis.

Treatment.—Energetic anti-syphilitic treatment is urgently demanded by every case of brain syphilis. This treatment is indicated even where there is merely a suspicion that a specific brain disease is developing. The most certain method is to give mercury at once, and to order inunction of 3·0 to 5·0 grammes of ung. hydrarg. per day, according to the urgency of the case. No objection can be made to the use of large doses in severe cases. I prefer the *inunction method* to any other mercurial treatment.

It is advisable to proceed in such a way that the inunction is applied to a different part of the body each day. We may begin for instance with the left arm, choosing the right arm the following day, then in the same way one leg after the other, and finally the back and thorax. The ointment must be rubbed in thoroughly and for a long time (about 15 to 20 minutes). The part of the body which has been rubbed should then be covered with a bandage. The patient may move about, if the symptoms are not of a threatening nature, and he may go out when the weather is fine. He should not be put upon low diet. He should gargle every hour with 4 to 5 per cent. solution of chlorate of potash, and should be careful to keep the mouth as clean as possible, as a severe stomatitis necessitates an interruption in the treatment and may thus be the source of grave danger. After the whole body has been anointed, the patient should take a warm bath or be washed. We need not here further discuss the other methods of treatment, such as the use of resorbin, Unna's mercurial ointment soap, the subcutaneous injection of various mercurial ointments and preparations, the use of the merco-lint bib, etc. It should certainly be admitted that subcutaneous or intramuscular injection of mercurial preparations has its advantages, and that there are certain conditions, *e.g.* abnormal constitution of the skin, etc., which necessitate its being adopted. Lewin, Jullien, Leredde, etc., consider it to be a better method than inunction in such cases. I have seen much benefit from enesol. It should also be said that the internal use of mercury has again lately been warmly recommended, and it is not always possible to avoid it. Subcutaneous injections of iodipin, advocated by Schuster and others, have proved successful in many of my cases, even in some in which other methods of specific treatment had failed. I have also used *iothion inunctions* with benefit. I have no personal experience of the intravenous introduction of preparations of iodide. The attempt to effect the direct action of specific drugs upon the central organs by means of dural infusion (Jacob) should find few imitators.

Although it is impossible to fix a standard, I think it advisable to rub in on an average at least 200 grammes (about 7 ounces) of grey ointment. If the severe symptoms have passed off, it is well to discontinue the treatment for a time, and to resume it after an interval of some weeks or months. Should the improvement be unsatisfactory, the treatment should be continued, but it seems to be advisable to interrupt it after a time, in order to watch the effect, which is often somewhat tardy. Generally, it is true, the results of antisyphilitic treatment soon become evident, commencing as a rule within the first or second week (Naunyn). If there is a relapse, the treatment should be again resumed. The patient should in many cases be advised to undergo this treatment once or twice every year. Fournier and Neisser, with whom Leredde, Lesser, O. Rosenthal,[1] and others agree, advocate intermittent treatment extending over many years. Iodide of potassium, in doses of 2—5—10 gr. per day (many physicians give even more), is a drug which is often wonderfully successful, but it is only in the slightest cases that one should depend upon it alone. It may be of special service in establishing the diagnosis *ex juvantibus*. Iodide seems in general to have the most rapid, and mercury the most lasting effect. Some objections have

[1] *D. m. W.*, 1904. See also the paper by Hallopeau and Lesser, *D. m. W.*, 1907, and Joseph, *ibid.*

been raised (F. Lesser) to the combined use of preparations of iodide and mercury, but they appear to me to be quite unfounded.

Subcutaneous or intramuscular injections of atoxyl in doses of 0·4-0·6 g. every second or third day, which have lately been recommended by Uhlenhuth-Hoffmann,[1] Neisser,[2] and others from the results of their experiments, have, in spite of a few favourable reports (E. Lesser, etc.), proved to be uncertain in their effects, and on the other hand they have in several cases produced severe and even irreparable changes, especially atrophy of the optic nerve (Bornemann, Fehr). In any case I would advise that in future experiments the dose should not be larger than 0·2 g. per day, and that the treatment should be suspended after 10 to 15 injections, the general condition being kept under strict control.

Antisyphilitic treatment is a complete failure in some cases. It may then be advisable to combine or to follow the mercurial treatment with a course of *hydropathy, baths, or a change of climate*. The baths of Aachen, Nenndorf, Weilbach, Tölz, Hall, Salsomaggiore, etc., have sometimes proved useful in this respect; a stay in the south, or mild cold-water treatment have also been successful in some cases. Neisser and others think, however, that sulphur baths should not be prescribed during the inunction treatment, as the sulphur neutralises the effect of the mercury. The baths are, however, compatible with subcutaneous or internal use of mercury. Simple sweating and Zittmann's treatment may also be recommended, but hot (and cold) baths should be avoided.

The paralytic condition calls for the use of electricity, massage, etc.

Surgical treatment has been adopted in some cases (Horsley, Macewen, Bramwell, Diller, Parker, Fischer, Nonne, Gajkiewicz, Rybalkin, Mills, Bayerthal,[3] etc.), either because a tumour had been diagnosed instead of the syphilis, or because antisyphilitic treatment had proved to be of no avail. Removal of gummata or callosities of the meninges in the area of the Rolandic fissure or the frontal lobe, was the usual operation in such cases. Horsley and Kocher are the strongest supporters of the necessity of surgical treatment of gummata, as they deny the efficacy of antisyphilitic treatment. Friedländer and Schlesinger[4] give the following indications for this treatment: It is suitable (1) when the symptoms of a tumour persist, in spite of antisyphilitic treatment, and point to a circumscribed focus accessible to operation; (2) when, in spite of antisyphilitic treatment, under the above-named conditions, the progressive course constitutes a danger to life; (3) when the symptoms of cortical epilepsy continue after the antisyphilitic treatment. I have never been able to decide to recommend operation in any case of cerebral syphilis.

Marie and Guillain recommend lumbar puncture for the headache which is due to syphilis. Donath has seen the other symptoms of brain syphilis pass off under this treatment in one case. Krönig[5] advocates this method most warmly in suppurative syphilitic meningitis.

We are justified in hoping that the future will bring us a method of *preventive inoculation* and of *serum-therapy* for syphilis.

[1] *D. m. W.*, 1907. [2] *Ibid.*
[3] See the bibliography and discussion in Stransky, *C. f. Gr.*, 1905.
[4] *Mitt. aus. d. Grenzgeb.*, iii. [5] *Dermat. Zentralbl.*, 1905.

In order to prevent relapses the patient must be urged to adopt a careful mode of life, to avoid all physical and mental exhaustion, emotion, the heat of the sun, trauma, and especially all excesses *in baccho et venere*.

The *prophylaxis* of cerebral syphilis, to which so many adolescents fall a victim, is practically identical with that of syphilis in general, and it is well here to point out how very thoughtlessly and with what a lack of experience even well-educated young men run the risk of syphilitic infection. I believe that much distress might be prevented by timely instruction and warning, What ethical training and self-interest fail to do might possibly be accomplished by fear of infection, if the laity were in a position to understand the whole extent and bearing of the danger. Although I am strenuously opposed to the medical advice which is disseminated by many of our daily newspapers and by modern literature, yet I regard it as necessary that young men, in especial, should be instructed upon these matters by their parents and teachers. Shortly after I had expressed this opinion (compare the first and second edition of this text-book), a beginning was made to carry these maxims into practice, but the movement should not cease there. It should, in any case, be regarded as an established fact that radical treatment of constitutional syphilis is the surest, though not an absolute protection from disease of the brain.

Progressive Paralysis of the Insane (Paralytic Dementia) [1]

This is a very common disease in civilised countries, and especially in large towns, and many individuals fall a victim to it in the prime of life.

Even in 1850, Moreau spoke of an increase in this disease, and there can be no doubt that it has become very much more widespread, especially within the last few decades (Wille, Régis, Snell, Thomsen).

Men of 30 to 40 years of age are chiefly affected, but the disease is not uncommon in women, and it would seem to have gained greatly in frequency among women during recent years (Griedenberg, Mendel, Jahrmärker). It may appear at the end of the second or the beginning of the third decade. Cases collected during the last twenty years, and steadily increasing, show that childhood is by no means exempt from this disease.

I had occasion when I joined the Charité in 1883, to treat and observe a case of this kind in a *girl of fourteen years of age*, but I was unable to make a definite diagnosis. Cases of this kind have since then been published by Régis, Wigglesworth, Bury, Strümpell, Clouston, Hüfler, Hirschl (*W. kl. W.*, 1901), Bresler, Mingazzini, Rad, Stewart, Haushalter, Alzheimer, Fröhlich, P. Bernhardt, Watson (*Arch. of Neurol.*, 1903), etc. The existence of a *juvenile* and *infantile* form of paralytic dementia has been proved by the post-mortem and anatomical examinations, first of A. Westphal, and then of a number of other writers (Haushalter, Raymond, Thomsen-Welsh, Toulouse-Marchand (*R. de Psych.*, 1901), Mott, etc.). The female sex is comparatively often affected. This condition has been very thoroughly studied by Mott (*Arch. of Neurol.*, 1899), whose work was based upon twenty-two cases of this kind. During the last few years Alzheimer has been able to extend his statistics to thirty-eight cases, and Fröhich (*Inaug. Diss.*, Leipzig, 1901) to eighty-three. See also Firpi, *Thèse de Paris*, 1906.

[1] Older literature in Mendel, "Die progr. Paralyse," Berlin, 1880; Krafft-Ebing, "Die progr. allgemeine Paralyse," Nothnagel's "Handbuch der Spez. Path.," etc., Bd. ix.; Binswanger, "Die pathol. Histol. der Grosshirnrindenerkrankung bei der allg. progr. Paralyse," etc., Jena, 1893.

Only a small proportion of the cases occurs during the sixth and seventh decades.

The opinion that *syphilitic infection* occupies the most important place among the *causes* of this disease has gained increasing acceptance.[1] This view, founded at first entirely upon statistics, gained a firm footing when Krafft-Ebing ventured to inoculate paralytics with syphilitic virus without thereby infecting them. Since then an increasing number of still more convincing facts have been brought forward which have caused the theory of the syphilitic origin of the paralysis to be almost generally accepted. First of all we had the important evidence of the results of examination of the cerebro-spinal fluid obtained by means of lumbar puncture; then the results of the investigations of Monod, Widal, Sicard, Joffroy, Mercier, Babinski, Nageotte, Anglade, Dupré, Nissl,[2] Brion, Meyer,[3] Siemerling,[4] Merzbacher,[5] Decoubaix,[6] Abraham-Ziegenhagen,[7] E, Schlesinger,[8] Fischer,[9] Liebscher,[10] Kutner,[11] etc., most of whom showed that the number of lymphocytes was *always* increased, a factor which could only be rightly interpreted when it was found that this pleocytosis is to all appearance found chiefly, if not exclusively in syphilis and syphilogenous diseases. Among the chemical reactions of the fluid, the increase in the amount of albumen seems to be of corresponding importance, as Sicard, Nawratzki, Marchand, Guillain-Parant,[12] Merzbacher, Schönborn, Henkel,[13] and others have concluded from their investigations. Nissl, however, did not succeed in obtaining any definite results, so that the value of this reaction seems doubtful. Nonne and Apelt[14] have lately made another careful test, and their experience of the fractional precipitation of albumin is that a positive reaction (marked increase of the albumin contents in the fluid) is only found in tertiary syphilis and syphilogenous nervous diseases, and always in paralysis. This was followed by the significant discovery of Wassermann,[15] Plaut, Citron,[16] and others of the syphilitic anti-bodies in the fluid and blood serum of individuals suffering from syphilogenous nervous diseases, and especially from paralytic dementia, or the serum reaction characteristic of syphilis and syphilogenous nervous diseases. It can, therefore, no longer be doubted that the *great majority of paralytics are subjects of syphilitic infection.*

Hereditary syphilis may also lay the foundation for paralysis, and it is apparently the usual cause of the infantile and juvenile forms of this disease. Raymond describes the combination of juvenile paralysis and cerebral syphilis. The comparatively frequent occurrence of paralysis in husband and wife (*conjugal paralysis*) (Kjelberg, Mendel, Mönkemöller[17]) also points to its syphilitic origin. The disease has only in rare cases been observed in mother and child or in children of the same family (O. Möller, Hoch, Klein, Gianelli, Oppenheim,[18] etc.).

[1] Of the later papers on this subject, see Fournier-Raymond, "Paralysie générale et Syphilis," Paris, 1905.

[2] *C. f. N.*, 1904. [3] *B. k. W.*, 1904; *A. f. P.*, Bd., xlii. [4] *B. k. W.*, 1904.

[5] *C. f. N.*, 1904. [6] Ref. *N. C.*, 1905. [7] *C. f. N.*, 1904.

[8] *D. m. W.*, 1904. [9] *D. m. W.*, 1904. [10] *W. m. W.*, 1906.

[11] *M. f. P.*, xviii. [12] *R. n.*, 1903. [13] *A. f. P.*, Bd. xlii.

[14] *A. f. P.*, Bd. xliii. [15] Comprehensive papers in *B. k. W.*, 1907, l., and *M. m. W.*, 1907.

[16] *D. m. W.*, 1907. [17] "Ärztl. Sachverst.," 1905.

[18] One patient brought his brother who was suffering from paralytic dementia to me, and from his speech, etc., I saw that he himself was suffering from incipient paralysis. A year later he

Fournier thinks that slight cases of syphilis, which have been insufficiently treated, are specially apt to become paralytic.

An interval of five to twenty years usually elapses between the infection and the onset of the paralysis—as the disease is usually inappropriately termed in practice.

But further, the etiological importance of *mental exhaustion, emotional excitement*, and especially of *excesses* should not be underestimated. Individuals who lead an agitated, restless, erratic, dissolute life run a special risk of acquiring this disease. Excesses *in venere et baccho*, especially chronic abuse of alcohol, undoubtedly aggravate the predisposition to this disease, although in such cases a number of harmful influences usually co-operate to produce it (Oebecke, Gudden). Krafft-Ebing has summed up the factors which have an etiological bearing under the term "*civilisation and syphilisation.*" These influences explain why artists, officers, stock-brokers, and merchants furnish the largest contingent to the disease.

Finally, many cases show that *injuries to the head* may be the exciting cause of general paralysis. The importance of this factor as a cause has, it is true, been very differently estimated by various writers, and has been altogether denied by some. Mendel [1] and Ziehen (as well as Fürstner) have expressed this opinion, but they have also admitted that when there is a pre-existing disposition (syphilis, etc.), trauma may be the exciting cause of the development of the disease. Thus Ziehen thinks that the syphilitic virus may settle in the brain after an injury has lowered its power of resistance.

Heredity is not an important factor in the etiology of paralysis. Naecke, however (and also Mariani, Schaffer, Joffroy,[2] and Raymond-Fournier), has lately stated with great emphasis that heredity (*i.e.* transmission of the hereditary degenerative disposition and the debility of the brain to which it gives rise) is of essential importance. These endogenous causes are the essential factors, whilst the exogenous causes, syphilitic infection in particular, are of secondary importance.

The theory of gastro-intestinal auto-intoxication (Bruce and Robertson, Macpherson) has little foundation. Nor can any great importance be attached to the bacteria found by a few writers (Robertson, *R. of N.*, 1903 and 1906).

Pathological Anatomy.[3]—General paralysis is mainly a disease of the brain, but the spinal cord is usually also involved. The cerebral changes in advanced stages are as follows:—The brain, in particular the cortex, appears to the naked eye to be *atrophied*. The *sulci* are *deepened*, the gyri small and narrow. The atrophy specially affects the *frontal* and *parietal lobes*, but other areas, such as the island of Reil and part of the temporal lobe, are also involved. The *pia mater* is often firmly adherent to the brain at certain areas, so that it cannot be separated without particles of cortical substance adhering to it; at a later stage, however, the pia may be easily separated. It is often thickened and

came under my care with the disease fully developed. I have since seen another case of two brothers with the same disease.

[1] *C. f. N.*, 1904. See also Gieseler, *A. f. P.*, Bd. xl. K. Mendel (*M. f. P.*, xxi.) also rejects the traumatic etiology.

[2] *Méd. mod.*, 1903.

[3] On this section compare Cramer, "Handbuch der path. Anat. des Nervensystems," Bd. ii., chapter 41.

opaque. *External hydrocephalus*, slight or marked in degree, is the usual result of the cortical atrophy. The contents of the dilated ventricle are also increased, and its walls are lined with granules (ependymitis granularis). Changes characteristic of internal hæmorrhagic pachymeningitis are not infrequently found on the dura. Morbid processes of an arterio-sclerotic nature are often present on the arteries at the base. The *weight of the brain* is usually greatly diminished, falling from about 1400 to 1000-900 grammes. This loss of weight affects mainly the frontal and parietal lobes. On section the grey matter of the cortex is diminished, possibly to a third of its normal breadth, but this is not always so. Binswanger distinguishes, according to the predomination of this or that change, three types of general paralysis, which he also endeavours to characterise clinically (see below).

Microscopical investigation of the brains of paralytics has yielded less definite results, but great progress has been made in this respect within the last few years, thanks to the investigations of Nissl and Alzheimer.[1] It is certain that the nervous elements undergo partial atrophy, but whether this atrophy is primary or whether the process arises from interstitial tissue (vessels, neuroglia) is not yet determined.

Many changes have been demonstrated in the nerve cells by the investigations of Binswanger, Meschede, Mendel, Mierzejewsky, Lubimoff, Tigges, Klippel (*R. n.*, 1903), Cramer, and very specially by those of Nissl and Alzheimer. The interstitial tissue and vascular system also take part in the morbid process. Thus we have mention of increase of the nuclei and fibres of the neurolgia, proliferation of the endothelial cells, infiltration of the adventitial sheaths, Marshalko's plasma cells, rod-cells and lymphocytes, dilatation and displacement of the lymph tracts, etc. (Magnan, Lancereaux, Mendel, Schüle, Recklinghausen, Raynaud, Wernicke, Joffroy, Elmiger, Nissl, Riss, R. Vogt, Mahaim, Cramer, Alzheimer, Buck, Klippel, etc.), and it has been inferred that the process is the result of a neuro-paralytic hyperæmia, leading to transudation with secondary lymph stasis, these again resulting in degeneration of the nervous elements. Binswagner, Orr, and Cowen, on the other hand, think the affection arises from the nervous structures. Alzheimer, who employed the new Weigert method, found glial proliferation extending over the whole cortex, *i.e.* over all the layers. He ascribes an important rôle to the inflammatory process, but thinks that the injury and destruction of the parenchyma is the most essential factor.

Investigations made with the methods of Golgi, Marchi, and Nissl, have not definitely decided the site of the origin of the process, although they point as a whole to its being of a parenchymatous nature. In any case the changes found in the nerve cells have no specific character, as is indicated by the investigations of Juliusburger and Meyer (*M. f. P.*, iii.) and the data of Cramer, and as Nissl himself has admitted. In spite of this, however, he regards the anatomo-pathological changes in general paralysis, taken as a whole, as thoroughly characteristic.

Tuczek[2] has demonstrated a fact of pre-eminent importance, viz., the *destruction* of the fine medullated fibres—the *tangential-fibres*—in the superficial layers of the cortex, especially of the frontal lobe (gyrus rectus) and the cortex of the island of Reil. K. Schaffer[3] concluded from his investigations that the process of degeneration affects preferably that zone of the cortex which Flechsig terms the association-fields (see p. 632), but he subsequently modified this view. According to the very thorough investigations of Kaes,[4] the atrophy of the fibres in the cortex is exceedingly diffuse.

[1] "Histol. Studien zur Differentialdiagnose der progr. Paralyse," Histol. und histopath. Arbeiten von Nissl, Bd. i., 1904.

[2] "Beiträge zur Anat. und zur Path. d. Dem. paral.," Berlin, 1884.

[3] *N. C.*, 1903.

[4] *M. f. P.*, xii.

Cramer concludes from all the evidence that almost every part of the brain may be subject to the paralysis, the cerebral cortex and the central grey matter on the floor of the ventricles being, however, most severely and most constantly involved. The atrophy of the medullated cortical fibres is most characteristic, but we have also to consider the condition of the vessels, especially of the adventitial spaces, the presence of endothelial proliferation, the emigration of leucocytes, and the accumulation of pigment in these spaces, and in the third place the condition of the cells and glia, of the ependyma, the soft membranes, etc.

In a few cases also (Rosenthal, Ascher, Lissauer, Boedeker, Sérieux et Mignot, Buder,[1] Hoch,[2] Pascal [3]), focal diseases have been found in the form of local softening, circumscribed meningo-encephalitis, or locally intensified degenerations, which have been regarded as an excess of the paralytic process, as Alzheimer in particular has lately shown.

Degenerative processes have also been found in the deep parts, such as the basal ganglia, and especially in the optic thalamus (Lissauer, Raecke), and have been explained as secondary degenerations. Siemerling [4] in particular has found focal diseases at these and other parts of the brain. The nuclei of the brain stem may also participate in the degenerative process, the nerves of the eye muscles being most constantly and most severely involved (Westphal,[5] Siemerling-Boedeker,[6] Juliusburger-Kaplan [7]), but cell changes have also been found, chiefly by means of the newer, finer methods of investigation, in the nuclei, especially the motor nuclei, of the medulla oblongata (Gerlach, Tolotschinow, Zitovitsch [8]), so that care must be taken when this condition is under consideration. We should here refer to the degeneration of the so-called bundle of Schütz (Schütz, Zitovitsch, etc.). The changes in the *spinal cord*, which is very often affected, demonstrated by Westphal's method, are of special interest. In some cases there is disease of the pyramidal tract, in others of the posterior columns, and in many, apparently the majority (Gross, Fürstner), both these systems may be involved.

The degeneration of the pyramidal tracts is regarded by Westphal as primary; Boedeker and Juliusburger considered it in one case as secondary, and Starlinger (*W. kl. W.*, 1899) has shown by his investigations that a secondary degeneration of the pyramidal tracts (and also of the anterior pyramidal) is produced by the cortical process in general paralysis. Fürstner (*N. C.*, 1900; *A. f. P.*, xxxiii.), who has studied this question with special care, also believes in the occurrence of this secondary degeneration. The degeneration of the posterior columns has been identified by many writers with that in tabes, whilst others differ in their views of its origin and localisation. We can only here refer to the investigations of Gaupp (*Psych. Abhandl. v. Wernicke*, 1898), of Taty and Jeanty, Heveroch, Sibelius, Wyrubow, Vigouroux, and Laignel-Lavastine (*Nouv. Icon.*, xviii.), and Kinichi-Naka (*A. f. P.*, Bd. xl.). Degenerative changes in the cerebellum have been described by Raecke, Binswagner, and by Borda (ref. *N. C.*, 1906).

It is impossible to discuss here the finer degrees of changes which have been described, *e.g.* in the neurofibrils of the ganglion cells, by Ballet (*R. n.*, 1904), Marinesco (*R. n.*, 1904), Schaffer (*N. C.*, 1906), and Bielschowsky-Brodmann (*Journ. f. Psych.*, v.).

Grey degeneration of the optic nerves has also been found, and less frequently a similar affection of the other cranial nerves. Recent investigations (Hoch, etc.) indicate that degenerative conditions also occur in the peripheral nerves in this disease.

For the changes in other (non-nervous) organs, see Pilcz, *Jahrb. f. Ps.*, xxv.

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[1] *Z. f. Psych.*, Bd. lx.
[2] *R. of N.*, 1907.
[3] *Thèse de Paris*, 1905.
[4] *N. C.*, 1899.
[5] Westphal-Siemerling, *A. f. P.*, xxii.
[6] *A. f. P.*, xxix.
[7] *N. C.*, 1899.
[8] *Dissert. Petersburg*, 1899; *N. C.*, 1900.

Symptoms.—The cardinal symptoms of this disease are the *progressive decline of the intelligence, the speech disturbance, the paralytic attacks, and the loss of the pupil reflex.*

Among these symptoms, the mental disorders occupy the foremost place. They are so very prominent that the disease has been rightly classed with the *psychoses.* Even before the mental disturbance becomes marked, subjective troubles and changes of disposition tend to become evident. These may in many respects resemble the symptoms of neurasthenia, viz., anomalies of mood, pressure in the head or headache, excitability, sleeplessness, giddy sensations, mental and physical fatigability, etc. Although these symptoms in rare cases show definite characteristics, *e.g.* sleeplessness marked by great persistency and resistance to treatment, sometimes by its combination with irresistible drowsiness during the day, etc., it is the *mental defect* which gives to the disease its peculiar stamp. The characteristic feature of this mental condition is the gradual weakening of the mental powers. The intelligence becomes evidently diminished during the first stages. The finer feelings, the ethical perceptions and sensations, are the first to become dulled. The patient makes use of expressions and performs actions which only close observers or those intimate with him perceive to be foreign to his nature. A serious man becomes frivolous, a considerate and decent man seems from some of his ways to have become rude or cynical. This mental defect soon manifests itself even in his outward mode of life, his dress, bearing, and manner. The work of deterioration begins first in the higher life of the mind and soul. His interest in his family soon grows weaker, he becomes callous or retains only the outward manifestations of feeling, whilst his power of love, friendship, etc., lose their depth and character.

About this time or soon after the power of perception becomes so greatly diminished that the patient grows incapable of any serious mental work, and the duties of his business are more and more neglected. A further advance in the impairment of mental capacity is shown if the *judgment* becomes so greatly diminished that the sufferer, hitherto of irreproachable conduct, commits some folly which brings him into conflict with the law. As a rule these acts betray not merely a *moral*, but also an *intellectual defect.* One individual, although well-to-do, will steal some trifle; another, who is happily married, will compromise himself with a courtesan, etc.

Should the disease in its initial stages have escaped recognition, it may suddenly manifest itself by some act startling in its singularity and depravity. One paralytic attempted to pass his excreta in broad daylight in the open busy street; another poured the ladies' champagne into their pockets; a third called out in the car, to ladies whom he did not know, to sit upon his lap; a rich man, in passing a butcher's shop, made off with a sausage, etc.

The *memory* is always and usually from the very first markedly impaired. The events of the immediate past become specially blurred; in other words the capacity to fix new impressions, to concentrate the attention, is usually most affected, whilst the memory of the far-away past may be long retained. In addition to this there is abnormal *excitability*; insignificant events may rouse immoderate excitement, which in its intensity is out of all proportion to the trifling cause. But the excitement does not last. It may rapidly pass into the opposite condi-

tion. On the other hand, the patient shows remarkable indifference to matters of essential importance.

Should we have no history of abnormalities in the family and social life of the patient, a short examination is usually sufficient to reveal the commencing dementia. His position, occupation, character, and sphere of interests in the past, must be taken into consideration. One should ask him to give a short description of his daily life, of the events of the past few days, and some question as to his business life should be put to him which necessitates an explanation. If he is likely to be well informed in politics or the most important events of the day, the examination should extend to these subjects. The mental impairment is usually markedly evident in the patient's inability to make easy *mental calculations*. The same requests should not, however, be put to every patient. Whilst difficulty in carrying out the mental multiplication of two double lines of figures may be a very serious matter when the patient is a book-keeper, a bank-clerk, etc., the fact that a working-man may not be able to do the simple multiplication-table may not be of any real importance. Above everything else one should always bear in mind the capacity of the person under examination. In advanced stages he may be unable to tell the year, his age, etc.

Testing with *association-stimuli* (Bleuler, etc.) may soon show the condition.

In examinations in which I desire to come quickly to the point, or to demonstrate the mental defect without delay to my students, I usually put the following questions: "What year is this?" Should the patient answer correctly, 1909 or so, I then ask, "What year was it five or seven years ago?" and in advanced cases I almost always receive a wrong answer, or I ask, "How much is seven times nine?" The patient answers correctly, "Sixty-three." "Then how much is nine times seven?" The patient considers a long time before giving a right or wrong answer. Heilbronner (*D. m. W.*, 1906) gives some useful hints for such examinations. Moreover, the mental dulness and vacuity become so soon evident in the *facial expression*, that there are few diseases which in their advanced stages one can so quickly diagnose at the first glance as general paralysis.

The dementia increases in some case rapidly, in others slowly, until the mind is entirely blank, and the patient becomes a complete imbecile. It is only in some cases—according to recent observations in the majority—that the mental trouble takes the form of simple *apathetic dementia*. It is often characterised by *hallucinations* and a *profound alteration of the emotional life*.

The hallucinations are often of the nature of *ideas of greatness*, and of a foolish and expansive exaggeration of self-importance. The patient considers himself a great artist, a discoverer who finds out something new and startling every day and hour, fancies he is God or the Kaiser, that he possesses countless millions, or that the whole world belongs to him. But these big ideas do not as a rule last; they are easily suppressed and replaced by others. The patient is incapable of being guided or of defending himself by a trace of logic. In almost as many cases the thoughts of the patient are governed by *hypochondriacal ideas*, and these may lead to great depression, especially if they develop at the beginning of the illness. The mental weakness is manifested early as a rule by the crass

absurdity of the ideas. The patient imagines he has no stomach or no bowels, or that his food goes to his brain; he thinks his head is too large or too small; fancies he cannot swallow or chew, etc. These ideas may at first be so powerful that the patient's general condition suffers, he loses strength, and may even die of starvation. But as a rule they do not last long and are replaced by ideas of greatness. At the onset of the disease the certain *consciousness of the approaching disease* may lead to profound melancholia.

Conditions of excitement are not uncommon at the commencement or during the course of the disease. They usually occur at an early stage, so that the condition may simulate *mania*. The patient is extremely restless, speaks constantly and at a bewildering rate; this gradually increases until the condition becomes one of wildest *frenzy*; he lays about him in blind rage, destroys everything within his reach, injures himself and others, uses abusive language, screams, howls, and pours out a stream of broken sentences, disconnected words, and senseless syllables. His features are contorted into grimaces, and as he usually takes no food, he quickly becomes emaciated. In many cases there is excessive *anxiety*. The more advanced the disease, the more evident is the mental failure.

These mental symptoms are usually associated at an early stage with *somatic signs of disease*. The chief of these is *loss of the pupil reflex*, which is not only a very frequent, but a very early symptom. It may indeed precede the development of the disease for many years and may, like the writing on the wall, foretell the future fate of the sufferer. The pupils are generally narrow, but mydriasis may be associated with the rigidity.

Westphal's students (Moeli, Thomsen, Siemerling, Wollenberg, Gudden) have studied the condition of the pupils in general paralysis in a large number of cases and have found loss of the light reflex in 50 to 60 per cent.; Siemerling even found this in 68 per cent., and Bumke (*M. m. W.*, 1907) in 87 per cent. of the cases. This symptom occasionally preceded all the others for five to ten years and more. It has also been pointed out that the pupils are often angular, indented and irregular in outline (Baillarger, Moeli, Musso, Salgo, Pilcz—the latter has carefully studied this symptom, see *N. C.*, 1903), and that the sensory reaction is also soon abolished (Moeli, Hirschl). See also Joffroy (*Arch de Neurol.*, 1904), Bumke, "Die Pupillenstörungen bei Geistes- und Nervenkrankheiten," Jena, 1904, and Bach's "Pupillenlehre" (1908).

Marked inequality of the pupils and "springing pupil" may be among the early symptoms. Paralysis in the external muscles of the eye and the accommodation muscles seldom occurs in the initial stage. It has been shown by the investigations of Westphal, Siemerling, Boedeker, etc., that opthalmoplegia may be one of the symptoms. Hemicrania with vibrating scotoma has more often been observed in the initial stage.

Another symptom of almost as much importance, and which we might indeed call pathognomic, is the *speech disturbance*—"the paralytic speech." It is specially characterised by the *pararthria syllabaris*, and by the dropping out of syllables, the tremor and shaking of the lips during speech, the associated movements in muscles of the face which are not normally involved in articulation (*e.g.* muscles of the forehead and ear), and finally by slight nasal speech. The pararthria syllabaris is the most significant of all. It is usually evident in conversational speech. In order to make it even more apparent, however, the patient should

be asked to say words or combinations of words which are difficult to pronounce, such as "artillery brigade," etc. It is also well to make him repeat the word several times, and it is peculiarly characteristic if the disturbance increases as he does so. The defective articulation is also very evident in reading aloud. It is obvious that the diction, or flow of talk, must suffer on account of the dementia. The speech disturbance may be the first symptom of the illness, but it is usually preceded by the mental anomalies. It is very seldom absent throughout the whole illness or until the later stages.

As the disease progresses, the power of *writing* suffers; it becomes tremulous, the various letters are very unequal, some strokes are too thick, others drawn out too far; then letters, syllables, and words are left out and doubled, or syllables transposed, etc. In the end the contents have no meaning whatever. A very peculiar kind of disturbance in reading, in the form of paralexia, has been described. The writing of healthy persons may of course show these disturbances in some slight degree, and it is therefore always advisable to compare the writing of the patient with something which he had written when he was in good health.

Facial asymmetry or unequal innervation of the two facial nerves is very often evident, even in the early stages. This gives rise to a lack of fineness in the movements of expression and to the tendency to associated movements, so that in opening the mouth, for instance, the forehead is wrinkled and the ears moved. This synergic innervation is specially marked in speaking.

The movements of the arms are also awkward and ungainly, although there is no real diminution in the strength with which they are carried out. The uncertainty and difficulty also affects the legs, and the gait is clumsy, heavy, and awkward. The impairment of the motility, however, does not amount to complete paralysis, apart from the attacks which we shall discuss later. On the other hand, paralysis of the peripheral type, especially peroneal paralysis, has been occasionally observed (Moeli, Pick), but it is merely a secondary, accidental symptom.

Tremor is usually present; it may be limited to some muscles or to one side of the body, or it may extend over the whole of it. It chiefly affects the muscles of the lips and tongue. The fibrillary tremor and shaking of the lips become specially evident in the attempt to speak, to show the teeth, or *protrude the tongue*. The latter movement is usually carried out in a very characteristic way: the tongue is *thrust out*, the mouth being widely opened, or it is repeatedly put out and drawn back again. The tremor in the extremities is sometimes very slight, sometimes so severe that it is a very prominent symptom. It is inconstant and irregular, the oscillations being moderately or very frequent. It specially accompanies movement, but it may be present during rest. Motor symptoms of irritation resembling those of chorea[1] and myoclonia may appear in the course of the disease. Catatonic symptoms have also occasionally been seen (Knecht, Séglas[2]). Reflex contractions in the muscles of the mouth may sometimes be elicited by stroking the lips, the hard palate, etc., and the mechanical excitability of the muscles of the face is also often exaggerated.

Symptoms of *tabes* are often associated with those of general paralysis. We need not here repeat what has been said as to the loss of the pupil

[1] See Dräseke, *M. f. P.*, xvii., with bibliography, etc. [2] *Nouv. Icon.*, xx.

reflex. *Atrophy of the optic nerve* sometimes appears, and it may be present for years before the mental changes develop. Westphal's sign, slight ataxia, Romberg's sign, bladder troubles, lightning pains, impairment of the sensibility, etc.,[1] are not uncommon. Indeed paralytic dementia may supervene upon a fully developed tabes dorsalis. This tabo-paralysis shows many peculiarities in its symptomatology and course (see below), and therefore forms a special type of general paralysis.

Symptoms of affection of the lateral columns, or of combined disease of the postero-lateral columns, are more frequent. They may even become evident during the first stage of the illness. The tendon reflexes are actively exaggerated and the motor power is somewhat diminished, but this condition very rarely amounts to spastic paraparesis or to paraplegia. Severe spinal symptoms of this kind precede the mental change only in a few rare cases.

We have still to mention the symptoms which appear periodically, and which are very seldom absent during the whole course of the disease, viz., the *paralytic attacks*. This name is given to attacks of various kinds, which may be divided into *apoplectiform* and *epileptiform*. They last as a rule from one or more minutes to half an hour and longer. The former consist of a state of confusion or loss of consciousness, lasting usually only for a short time, and followed by paralysis in the form of hemiplegia, monoplegia, or aphasia. This paralysis lasts only for a few hours or days and then disappears. Babinski's sign is often present on the affected side during the attack (Robert-Fournier). An apraxia may also develop in the same way (Abraham, Lewandowski). Persistent paralysis of this kind is very rare (it has been observed by Ascher, Rosenthal, Moratow, Sérieux, Pascal, and Bumke). The temporary paralysis often follows a slight faint, or even an attack of vertigo. There may, however, be no impairment of consciousness, the attack being represented by a rapidly passing paralysis, or even by aphasia which lasts only some minutes. Such attacks are apt to return and usually to leave behind them some deterioration in the general condition, especially in the mental state and the speech.

The epileptiform attacks correspond either to *petit mal* or to true epileptic attacks (there are cases of paralysis in which attacks of this kind long preceded the onset of the disease), or much more often to the cortico-epileptic attack in its motor or sensory form. Unilateral convulsions are most common; paræsthesia, with or without loss of consciousness, is less frequent. There may be a succession of such attacks resulting in true status epilepticus. Paralytic attacks of purely psychical nature have also been mentioned, and the conditions of sudden and rapidly disappearing confusion and excitement have been included with these.

The attack of paralysis sometimes leaves behind it a *visual disturbance* corresponding to the type of mind-blindness (Fürstner, Stenger). It is usually of short duration.

We have no certain knowledge as to the cause of these attacks. It has been assumed that circulatory disturbances, small hæmorrhages, local œdema, or even a local aggravation of the underlying pathological process (encephalitis ?) may give rise to the symptoms. Some writers,

[1] I have seen a case in which gastric crises were for years the only symptom which succeeded the development of a paralytic dementia. The Achilles jerk often disappears before the knee-jerks. The special data given by Pilcz (*N. C.*, 1906) as to the condition of the sensibility in the early stage of the paralysis require further control.

such as Pierret and Donath, attribute the attacks to auto-intoxication. It has even been thought possible that peripheral stimuli (an over-full bladder, etc.) may act as an exciting factor. Starlinger (*W. kl. W.*, 1899) in particular has succeeded by means of Marchi's method in detecting changes corresponding to the focal symptoms, if death has followed rapidly upon the attacks. These showed, in particular, degeneration of the pyramidal tracts, which he regarded as secondary, and ascribed to the cortical affections underlying the attacks. See also Bumke (*N. C.*, 1904).

The temperature is raised during the attacks, and it may even be so in the intervals between them.

Pappenheim (*M. f. P.*, xxi.) found increase of the polynuclear leucocytes in the blood and fluid in these paroxysmal febrile conditions. See also Sorokowikow, *Dissert. Kasan*, 1904; ref. *N.C.*, 1905.

Vasomotor and *trophic* disorders (perforating ulcers, spontaneous fracture, arthropathy, gangrene), disturbances of nutrition in the nails, hair, etc. (Cololian), are not uncommon in paralytic dementia.

Zahn (*N. C.*, 1906) describes acute vesicular peeling of the skin.

Pruritus may be one of the symptoms, but it occurs under so many other conditions that no importance can be attached to it. This is true also as regards salivation. Niessl (*B. k. W.*, 1902) thinks the appearance of cyanosis of the face, especially of the eyelids, is of some importance. "Trichotillomania" (Féré), *i.e.* the tendency to pull out the hair, is not a special symptom, but it is often shown by the inmates of an asylum for the insane.

Course of the Disease.—The illness has usually an insidious development, and is protracted in its further course. It may happen, however, that the symptoms, hitherto slight, may become acutely and markedly intensified, especially after a paralytic attack. *Remissions* are not unusual: the symptoms may so completely disappear that the patient seems to the laity to be cured, and he is often able once more to resume his business. But this recovery is almost always deceptive. After a few months, possibly six, but only in rare cases a longer interval (two, three, and in very exceptional cases even six years), the relapse occurs. In two or three years after the onset, the mental weakness has usually developed into imbecility, and the speech disturbance has become so great that the patient cannot make himself intelligible. In the end speech may be absolutely impossible. The expression of the face reveals the complete mental deterioration. Walking becomes more and more difficult and finally impossible. The patient passes his urine and fæces unconsciously, becomes very unclean, etc.

At this stage or even earlier the feed-reflex described by Wagner may sometimes be elicited, the patient opening his mouth wide, like a young bird, and snapping whenever an object of any kind (key, match) is brought close to his face (see Dohrschansky, *Jahrb. f. P.*, xxviii.). This is a sign of profound imbecility.

Death occurs within the first two to three years. There is also a *galloping* form of general paralysis, which ends fatally within a few weeks or months. This form has been specially described by Buchholz [1] and Weber.[2] On the other hand the illness may even be prolonged for as many as ten years, and a few rare cases have been described in which it lasted for twenty years (Lustig, Schäfer). The course is generally slower in women. It also seems to be more gradual in the form associated with dementia and in tabo-paralysis than in those in which there is pro-

[1] *A. f. P.*, Bd. xxxvi. [2] *M. f. P.*, xiv.

found depression and violent exaltation. Senile paralysis is also slow in its progress (Alzheimer, Gaupp).

Binswanger describes three kinds of course, and connects each of them with the pathological changes: 1. The meningitic-hydrocephalic form, which shows a typical remitting character; health apparently quite good during the remissions; attacks with deep loss of consciousness, symptoms of spasm and paralysis, dementia increasing with the number and duration of the attacks. Pathology: extensive atrophy of the brain, enormous dilatation of ventricles, etc. 2. The hæmorrhagic form, with acute onset of the most severe symptoms (deep stupor, conditions of hallucinatory excitement, unilateral motor symptoms of irritation), rapid mental deterioration. Resemblance at the height of the disease to toxico-infective brain affections. Pathology: a considerable number of miliary hæmorrhages in addition to the well-known diffuse changes. 3. The tabo-paralytic form, preceded by tabes. Protracted course, extending often over ten years; prolonged arrests. The marked moral defects are often associated with wonderful intelligence, etc.

Lissauer, Alzheimer (*N. C.*, 1902), and others distinguish between *typical* and *atypical* paralyses, and attribute the clinical peculiarities to the special localisation of the process within the brain. The typical forms are localised chiefly in the anterior segments of the cerebral cortex; the atypical forms arise from the localisation of the lesion in the posterior segments of the cerebrum, in the optic thalamus, or from the initial involvement of the cerebellum, etc.

Death is caused by inanition, broncho-pneumonia, bed-sore, cystitis (in rare cases by spontaneous rupture of the bladder [M. Edel]), or by some intercurrent disease. It occurs less frequently during a paralytic attack or in the status epilepticus.

My experience agrees with that of Oebecke, Mendel, Fürstner, Schaffer, Näcke, etc., who think that the description of the course of the disease, founded upon earlier observations, can no longer be generally applied, or else that we now see a great many cases which essentially deviate in symptoms and course from the type first described. I have frequently observed remissions lasting for one to two years. Further, the mode of development of paralytic dementia in many individuals suffering from tabes has seemed to me to be quite atypical; the symptoms appeared suddenly and disappeared just as rapidly, giving place to a mental condition which apparently remained quite normal, until the dementia returned six months or more later. In several cases this transformation took place so rapidly that the patient, who had been confined as a lunatic, was released after a few days or weeks. In one case the disease commenced with the symptoms of severe hypochondriacal melancholia; the patient was taken to an institution from which he escaped, and in a short time he recovered and was able for a whole year to attend to his business as an apparently healthy man. In some other cases the patient has committed some single act which definitely pointed to the existence of mental disturbance, but for the space of six months to a year the most close and careful examination may fail to show any other sign of mental change, or of defective intelligence. Some patients (tabo-paralytics) have surprised me by the clearness and precision with which they express themselves, orally and in writing, and defend their interests during these periods of intermission. I have also been astonished to find that in some cases I could detect no speech disorders of any kind until the last stages of the illness.

The peculiar course in so-called tabo-paralysis has also been described by Binswanger, Fürstner,[1] Torkel [2] and others. Other writers, such as

[1] *M. f. P.*, xii.

[2] *Inaug.-Diss.*, Marburg, 1903.

Jolly and Gaupp, refuse to admit that the clinical picture of the disease has undergone any change in the course of the last ten or twenty years. In any case one cannot be too careful in avoiding any fixed idea of a definite morbid scheme of paralytic dementia, as this disease presents every imaginable variety of symptoms and course. This fact is of great practical interest. The physician who certifies a patient of this kind to be detained in an asylum must be prepared to find that after a short time even the most marked mental symptoms may have subsided, etc.

The *prognosis* is extremely bad. Every alienist of much experience is certain to know one or two cases in which he has diagnosed general paralysis and seen his patient recover, but these cases form a vanishing minority, and it is always possible that the diagnosis may have been at fault. Krafft-Ebing states that out of 2500 cases he has never seen one recovery, but he quotes a case of Svetlin's which hardly leaves any doubt as to the possibility of this occurrence. Fürstner had three or four cases of this kind in his own experience. Halban[1] has also reported cases of recovery, whilst Gaupp[2] says he has never seen a patient recover from true general paralysis. The remissions often follow an intercurrent febrile disease, in particular, erysipelas (Steiner,[3] etc.), typhoid (Foerster[4]), pneumonia, or profuse suppuration.

As regards the so-called stationary paralysis, see Wickel (*C. f. N.*, 1904), Obregia-Antonin (*Spitalul.*, 1906), Doutrebente-Marchand (*Ann. méd. psychol.*, 1903).

Diagnosis.—Paralytic dementia is a disease with which every physician should be most thoroughly acquainted in order that by a timely diagnosis he may prevent much family unhappiness. This is certainly not always an easy matter. The difficulties may be especially great in the *initial stage*. The first symptoms may be closely allied to those of *neurasthenia*. In several cases of this kind I had at first diagnosed neurasthenia, and only after the lapse of a year or even after several years I was compelled to admit the presence of general paralysis (the reverse was extremely rare). In such cases one should first inquire into the symptoms which are not characteristic of neurasthenia; these include first of all the loss of the pupil reflex or very sluggish reaction of the pupils (" springing pupil " apparently occurs in very rare cases of neurasthenia also), Westphal's sign, and paralytic speech and writing. It should not be forgotten, however, that neurasthenics in a condition of excitement or after prolonged insomnia sometimes show a disturbance of speech resembling pararthria syllabaris. But in such cases the symptom is transient and inconstant, and disappears when the test is repeated several times.

From my own experience I am extremely doubtful as to the statement of Pilcz that loss of the pupil reflex may be a transient occurrence in neurasthenia, whilst the cases of Cramer, H. Vogt, and Weber,[5] which show that immobility of the pupils appears as a transient symptom in degenerates under the influence of alcohol, deserve to be taken into consideration.

When these symptoms are absent, careful consideration of the mental condition is usually sufficient to confirm the diagnosis. The neurasthenic frequently complains of his loss of memory and of mental power, but this is practically caused by mistaken and morbid judgment of his own con-

[1] *Jahrb. f. P.*, xxii. [2] *D. m. W.*, 1904. [3] *W. kl. R.*, 1906.
[4] *M. f. P.*, xvi. [5] *M. f. P.*, xxi.

dition. When the diminution of intelligence, the changes in character above described, and the weakness of memory are objectively evident, the case is one, not of neurasthenia, but of commencing general paralysis of the insane.

The neurasthenic understands his condition to a certain extent; he gives a clear, detailed description, from which it is clearly evident how sharply he watches himself, how he tracks out every sensation, and how exactly he has followed the development of each pathological process. It is, therefore, generally, though by no means always evidence against the existence of general paralysis, when the patient is himself afraid of falling a victim to it. In the descriptions given by the paralytic his apathy, indolence, and defective memory usually become very soon apparent. There are cases of this kind, on the other hand, in which the feeling of illness is very acute; it oppresses and absolutely overwhelms the patient, producing a profound and lasting despondency, which is hardly ever observed in neurasthenia.

Imperative ideas point in doubtful cases to neurasthenia, even when they are absurd in their content. but I know one case in which a congenital neurasthenia with such ideas was associated later with general paralysis.

Examination of the fluid obtained by lumbar puncture has lately acquired great importance as regards the diagnosis. We have referred already to the numerous examinations which have yielded more or less consistent results as regards lymphocytosis, hyperlymphocytosis or pleocytosis. The results of chemical examination of the cerebro-spinal fluid, especially the increase of albumin, and the great importance of Wassermann's serum reaction, have also been already considered. These criteria are of special value in cases in which the difficulty of making a differential diagnosis between neurasthenia and paralytic dementia is peculiarly great. They only prove, however, that the individual has suffered from syphilis, and that it is, therefore exceedingly probable that the existing nervous disease is of syphilogenous origin. One should not go any further in one's conclusions.

The not uncommon combination of tabes dorsalis with neurasthenia may give rise to great difficulty in differential diagnosis, and here also, as in serum reaction, nothing is definitely proved by positive cytological and chemical results of examination of the fluid. We do well in cases of this kind to give the diagnosis of general paralysis only on the ground of *increasing* mental disturbances or of the presence of paralytic symptoms which are foreign to tabes (paralytic speech).

The paralytic attacks—when they are *typical*—may in doubtful cases be a valuable guide to the diagnosis, but unilateral convulsions, attacks of vertigo, and temporary inhibition of speech occur also in hysteria and hystero-neurasthenia. We should also remember that conditions very closely allied to these paralytic attacks may appear in association with hemicrania.

Among the other diseases of the nervous system with which paralysis may be confused, we should specially mention *cerebral syphilis* and *disseminated sclerosis*.

Gummatous meningitis, when it involves the region of the speech centre and the motor zone, may give rise to attacks of transient paralysis of the speech and extremities, and to convulsive attacks which are practically identical with those of general paralysis. But as a rule there is also headache, usually felt at certain sites, and often local tenderness

of the skull to percussion; and although the paralytic symptoms may at first tend to disappear, a certain amount of paresis persists even in the intervals between the paroxysms. The development points, in short, to a focal disease. The characteristic pararthria syllabaris is peculiar to general paralysis, whilst in syphilis the speech disorders are of another character (aphasia, bulbar speech, etc.). Optic neuritis is evidence in favour of a specific disease, whilst simple atrophy of the optic nerve decides in doubtful cases in favour of paralysis. Treatment may also throw light on the question, as antisyphilitic measures are as a rule only successful in true syphilis, and of no avail in general paralysis. Naturally spontaneous remissions may coincide with the period of treatment and add difficulty to the diagnosis. The most essential element of the mental disturbance in cerebral syphilis is the periodic stupor; in general paralysis it is the dementia. There is, it is true, a dementia associated with syphilis (a post-syphilitic dementia), but it is not progressive and is seldom developed to a high degree, so that the patient for a long time retains an insight into his condition (Winkel, Klein). The long prodromal neurasthenic stage is also absent in cerebral syphilis, as Patrick [1] has rightly pointed out. Cases occur, nevertheless, in which the most experienced observer cannot come to a definite decision. I have found it impossible, in one case which lasted for a year, to be certain of the diagnosis; and in another, in which on my first examination I gave the probable diagnosis of tabes dorsalis, I remained for the following year and a half utterly uncertain in my judgment, until at the end of that time unmistakable signs of general paralysis made their appearance. In the interval the patient wrote letters which were quite astonishing in their clearness and ease of diction. There is in particular a form of diffuse syphilitic meningoencephalitis, which cannot always be clearly distinguished from paralytic dementia. Even pathologically [2] it may be very difficult to differentiate between them, but Nissl and Alzheimer [3] have collected some important indications for this diagnosis. French authors (Fournier, etc.) in particular speak of syphilitic pseudo-paralysis; they maintain the existence of numerous intermediary conditions between it and true paralysis. Fürstner also maintains the possibility of pseudo-paralysis. Jolly [4] would limit the term syphilitic pseudo-paralysis to cases which correspond clinically to the picture of paralysis, and in which pathological examination shows, in addition to the diffuse degenerative processes, true syphilitic changes in the brain. A. Westphal has described very interesting cases of cerebral syphilis which resembled paralysis in the maniacal character and the ideas of greatness, but differed from it in the absence of dementia and the stationary character of the disease. Alzheimer also speaks of transition forms. I think it possible that the paralysis may in many cases develop out of a true cerebral syphilis.

Some of the criteria just given apply also to the differentiation of cerebral tumour and paralytic dementia (see also p. 920). Tumours of

[1] *N. Y. Med. Journ.*, 1898.

[2] Compare Pavlekovic-Kapolna, *Thèse de Lausanne*, 1903; ref. *N. C.*, 1903; Dupré-Devaux, *R. n.*, 1905, etc.

[3] *Loc. cit.*, and *C. f. N.*, 1905. See also E. Meyer, *A. f. P.*, Bd. xliii.; Ris, "Corresp. f. Schweiz. Ärzte," 1907.

[4] *B. k. W.*, 1901. See also Rentsch, *A. f. P.*, Bd. xxxix.; Marchand-Ollivier, *Rev. de Psych.*, 1904; Sträussler, *M. f. P.*, xix.

the frontal lobe and third ventricle have specially given rise to repeated confusion. In doubtful cases the presence of hyper-lymphocytosis of the cerebro-spinal fluid and Wassermann's reaction may determine the existence of general paralysis.

As regards the diagnosis of paralytic dementia from disseminated sclerosis, we would refer to p. 341. According to Tigges, Claus, Schultze and Zacher, Fürstner, and others, there is also a mixed form of these two conditions, but this co-existence must be extremely rare. Cytodiagnosis to all appearance affords no guide for the differentiation between disseminated sclerosis and paralytic dementia.

There is a brain disease of a diffuse kind, due to *arteriosclerosis*—an atrophy which in many points resembles paralytic dementia—but in such cases the mental disturbances are almost always associated with persistent symptoms of paralysis (hemiplegia, dysarthria, dysphagia, etc.) arising from the focal disease. The dementia is hardly ever so great as in general paralysis, and the speech disorder is of another character. This form resembles *senile dementia*, which, however, as a rule only occurs after the age of sixty. Alzheimer and Binswanger have carefully studied these morbid conditions, especially with regard to the differential diagnosis.

In senile dementia the physical signs of general paralysis are absent; the pupils are usually narrow and sluggish in their reaction, but the pupil reflex is very seldom lost (Moeli, Siemerling, Heddaeus, Bumke), and there is no pararthria syllabaris.

Binswanger also describes a chronic progressive subcortical encephalitis, in which the white matter is said to be atrophied chiefly in the posterior segments of the brain, but the process seems to have a merely pathological interest.

It is also uncertain whether the *colloid-degeneration* of the brain described by Alzheimer can, owing to the small number of cases, be differentiated from general paralysis, although in one of his cases the clinical picture differed essentially from that of paralysis and suggested cerebral tumour. In isolated cases hæmatoma of the dura mater has produced a clinical condition very like that of paralytic dementia.

Finally, *alcoholism* may give rise to symptoms some of which are allied to those of paralytic dementia. These include the tremor, motor excitement, and speech disturbance. When alcoholism produces these symptoms, it as a rule gives rise at the same time to delirium tremens, and this so little resembles the mental condition of general paralysis, or even the conditions of excitement in this disease, that the differentiation can in most cases be easily made from the symptoms. The psychosis which accompanies alcoholic neuritis certainly indicates a mental weakness, but it can as a rule be distinguished from general paralysis by the numerous illusions and hallucinations, by their typical character, and by the other signs of alcoholism. Moreover, the reflex immobility of the pupils is practically always absent, and the sluggish reaction usually disappears during abstinence. (According to Bumke, on the other hand, sluggishness of the reaction of the pupils and absolute rigidity are not uncommon in chronic alcoholism.) Forms of alcoholic dementia also occur which correspond so entirely to general paralysis that the authors think themselves justified in speaking of an alcoholic pseudo-paralysis (Marandon de Montyel, etc.). Gaupp has also opposed the standpoint which I took in the second edition of this text-book. It is still uncertain, though it seems probable, that cytodiagnosis may decide this question. Bruns reports a case in which *urœmia* simulated a paralytic condition;

Laudenheimer describes diabetic pseudo-paralysis, of which Ingegnieros (*R. n.*, 1905) also speaks. It has already been noted (p. 823) that transient hemiplegia, etc., may occur in diabetes. In disease of the liver a clinical condition may also develop which has a superficial resemblance to that of paralysis (Joffroy). A gouty pseudo-paralysis has also been mentioned (Klippel, Conso), but I have never met with it. Chronic lead-poisoning may give rise to symptoms very similar to those of general paralysis, but which sometimes terminate in recovery (Régis, Ball, Oppenheim). Similar conditions have also been observed in poisoning by bisulphide of carbon (Laudenheimer, G. Köster). Symptoms may occur in chronic bromide and trional poisoning which have certain features in common with paralysis, but these conditions are easily recognised from their etiology. It may be more difficult to differentiate between morphinism and paralytic dementa; indeed, it would seem as if the chronic use of morphia may produce a form of paralytic dementia.

This is not the place to discuss the differential diagnosis between paralytic dementia and mania, melancholia, etc., but it should be remembered that each of these psychoses, mania especially, if it appear in a hitherto healthy man, must arouse the suspicion of general paralysis. The physical symptoms or mental weakness usually enable the condition to be definitely recognised. Melancholia, or severe hypochondria, which develops to a certain extent abruptly in an adult man, who has hitherto been healthy and not nervous, is suspicious. Paralytic dementia seems, in contrast to this psychosis, and also to dementia præcox, to be well characterised by increase of lymphocytes.

It is sufficient here to refer to the hereditary family diseases which are characterised by the combination of progressive dementia with spastic symptoms (Homén, Trénel, etc.), without going into a discussion of their differential diagnosis. Whether a special form of dementia described by Turner can be distinguished from progressive paralysis can only be shown by further experience. This is the case also as regards the type described by Probst.

Treatment.—It is true that it is hardly within the power of the physician to cure this disease, but he may by suitable and timely measures avert many causes of injury and create the conditions most favourable for improvement. In the first stage, when the patient, still free to follow out his business and his social life, may, on account of his commencing ideas of greatness and the dulling of his moral sense, do the greatest harm to himself and his family, there is usually urgent necessity for confining him in some institution. This is not merely a protection for the patient, but it saves him from many irritations to which he is subject outside. We must remember, however, that the severe symptoms may rapidly subside (see above). It is usually unwise to yield to the wishes of the family or the patient himself that he should be taken to a hydropathic or institution for the treatment of nervous cases; the general paralytic in the acute stage of his illness should be in a *closed institution*. This confinement is not necessary so long as the only symptoms are the physical signs and simple apathy or weakness of memory, but one must always be prepared for the sudden onset of marked disturbances, and one should especially be careful never to let the proper moment slip for installing a nurse or putting the patient under legal guardianship. In the later stages, when the dementia is often far advanced and the condition of excitement hardly ever appears, the patient may be allowed to live with his family under constant, careful supervision.

The beneficial effect of rest in bed and nourishing diet on the course has been lately emphasied by Kaes.

As regards *direct* treatment, it has certainly seemed wise to adopt antisyphilitic measures, and this treatment has always been recommended. Unfortunately the results are so discouraging that many physicians have become directly opposed to it. I still think we are justified in prescribing energetic inunction treatment in fresh cases in which syphilis has undoubtedly been present, and especially in atypical cases in which there is even a possibility that we are dealing with a syphilitic pseudo-paralysis. The internal use of iodide of potassium or subcutaneous injections of iodipin may be tried in every case. The combination of mercurial treatment with the use of thyroid preparations is recommended by Splenger and others, but we are justified in viewing this treatment with grave doubts.

We need not here enter into a discussion of the attempts at serumtherapy (Robertson and Douglas M'Rae, *R. of N.*, 1907).

For the insomnia, preparations of bromide may be tried, and should these fail, sulphonal, trional, veronal, and morphia. Chloral hydrate should not be used as a narcotic, at least in the initial stages.

For treatment of the conditions of excitement, hyoscine, in addition to rest in bed, has been most successful, but in such cases it must be given in full doses.

In the paralytic attack the application of the ice-bag to the head, and especially the use of chloral and amyl-hydrate per rectum, should be recommended. A threatened attack may sometimes be warded off in this way. We should not forget that the bladder should be emptied.

A mild hydropathic course may be advantageous in the first stages, and in any case it does not tend to do any harm. Too energetic courses of treatment, especially the use of cold-water douches, should be avoided.

Some alienists advocate warmly the use of derivative methods, especially the application of the ung. tart. stibiat. to the shaven skull. It is also maintained that the prolonged use of an ice-bag has been followed by success. Donath thinks that transfusion of common salt has had a good effect.

The sitophobia can only be successfully overcome in an institution, as forced feeding may eventually be necessary.

Appendix

This includes the cursory discussion of some morbid processes which hitherto have had merely a pathological interest, as they have shown no relation to any definite recognised disease or have been insufficiently reported and classified. These represented partly accidental post-mortem findings, and partly changes which were occasionally found in paralytic dementia, *idiocy*, or other conditions

Under this category I include the brain affection described as *diffuse sclerosis*, in which the whole brain or large parts of it are said to be indurated, firm, leathery, tough, etc. The essential points as to this disease have been mentioned on p. 344. These changes are found under very varying conditions, and they therefore differ as regards their mode of origin and nosological importance. General induration and atrophy is a common condition in the *idiot* brain. This is not the place to discuss the pathological anatomy of idiocy or of microcephaly, but we may point out that, in addition to the changes already described, malformations have also been found, *e.g.* defects or distortion of whole segments or of some parts, such as absence of corpus callosum, asymmetries, anomalies in the arrangement of the convolutions, malformations such as microgyria (p. 841), macrogyria, heteropia, *i.e.* localised accumulations of grey matter

in the form of nodules at certain sites, which do not exist in the normal brain (Virchow, Simon, Tüngel, Otto, Campbell, etc.), hypertrophy caused by a local increase of glial tissue, focal diseases, etc. There can therefore be no suggestion of a uniform pathological process, and on the other hand, many of these changes are occasionally found in non-idiot brains. Shuttleworth-Beach (*N. C.*, 1900) and Bourneville have lately thoroughly studied the pathological basis of idiocy, and have drawn attention to the variety of the morbid processes. The histological condition of the cortical cells in idiocy has been specially described by Hammarberg ("Studien über Klinik und Path. d. Idiotie," Upsala, 1895), whilst Kaes (*M. f. P.*, i., with bibliography) has shown by his examination of the microcephalic brain that the development of the medullated fibres of its cortex is markedly behind that of the normal brain. A comprehensive description is given in the paper by E. Schütte (*Centralbl. f. allg. Path.*, 1900). Weygandt (*C. f. N.*, 1905) attempts to classify idiocy on the basis of the pathological changes. See also his report at the Jahresversammlung der Psychiatrie, Halle, 1906.

We have already had occasion to refer to diffuse sclerosis in the chapter on *infantile cerebral paralysis* (p. 841), in connection with those cases in which the diffuse sclerotic process follows a localised disease of the brain (Andral, Cotard, Jendrássik, Marie, Bischoff). It is one of the peculiarities of the localised brain diseases acquired in early childhood that they give rise to the development of a diffuse inflammatory process in their immediate neighbourhood and even in more distant parts. Wernicke regards this affection as a kind of softening peculiar to fœtal life and childhood, in which the necrosis attacks the nervous parenchyma alone, the glial tissue remaining intact. This process undoubtedly occurs sometimes in adults also, as is specially shown by the digest of Cotard.

Campbell (*Br.*, 1905) has recently published a comprehensive review of diffuse cerebral sclerosis.

There is even more doubt as regards a number of other conditions, which have hitherto been regarded as of hardly any clinical importance. These include so-called *tuberose sclerosis* (and gliosis), *i.e.* the development of nodules and larger nodes on the surface of the brain, due to local proliferation of the glial tissue in idiocy, epilepsy, senile dementia, and other conditions (Cruveilhier, Bourneville, Brissaud, Koch, Brückner, Fürstner, Stühlinger, Buchholz, Scarpatetti, Sailer, Philippe-Hudovernig, Ugolotti). See also Pellizzi (ref. *N. C.*, 1903), Perusini (*M. f. P.*, xvii.), White (*R. of N.*, 1905), and Geitlin, "Zur Kenntnis der tuberösen Sklerose," Berlin, 1906.

Senile sclerosis of the cerebral cortex is mainly due to arterio-sclerotic degeneration of the cortical vessels. A perivascular gliosis also occurs (Alzheimer). Spots of *vitreous degeneration* in the cerebral cortex have also been described (Schüle, Simon, Holschewnikoff, Warda).

Ripping found *cystoid degeneration* of the cortex in the form of cysts the size of the head of a pin in paralytic dementia. Fürstner mentions a disseminated formation of cavities due to other causes. Warda speaks of multiple, colloid degeneration of the cortex in one case, and Kazowski mentions multiple necrosis.

Tuke and Gowers describe *miliary sclerosis*; the former was found in the white matter of an atrophied cerebellum in a case of mental disease. Gowers, who quoted this case, saw in another, small reddish-grey foci from the size of a dot to that of a mustard-seed scattered over the brain, the cortex, and the central ganglia. There was nothing characteristic in the symptoms, but syphilis had been present. Similar changes occur in paralytic dementia also (Greiff), and Redlich has described analogous conditions.

We can merely refer to the very interesting studies on hemicephalus, anencephalus, hydrocephalus, and other malformations by Cruveilhier, Virchow, Monakow, Flechsig, Leonowa (*A. f. P.*, Bd. xxxviii.), Veraguth, Muralt, Sternberg (*Z. f. N.*, Bd. xxxiv.), Wichura, Ilberg, Petrén, Muscatello, Brissaud-Bruaudet (*Nouv. Icon.*, xvi.), Vaschide, and Vurpas, etc.). The communication of Probst (*A. f. P.*, Bd. xxxviii.) is one of the most recent, and Monakow's school has notably enlarged our knowledge of these subjects through the investigations of H. Vogt (*M. f. P.*, xviii., and *Arb. aus Monak. Inst.*, 1905).

DISEASES OF THE PONS AND MEDULLA OBLONGATA

The most important pathological changes have already been described on pp. 638 to 646, and pp. 656 to 665, and we would again refer to the corresponding figures and the whole description there given of its anatomy and physiology.

In order to understand the pathology it should be clearly understood from study and tracing out of the various tracts and nuclear groups, that the fibre masses containing the *motor and sensory conduction tracts* are here compressed into a narrow space, which moreover contains the *nuclei of the cranial nerves*, with the exception of the first, second, and third, arranged symmetrically.

Although there is no sharp division between the pons and the medulla oblongata, it is expedient from a nosological point of view to draw such a dividing line, and to regard as belonging to the pons the portion which at the basal surface is covered by the so-called stratum superficiale pontis. A glance at Figs. 282-288 shows that the pyramidal tracts are contained in the basal or under portion of the pons, and are in the proximal levels split up by the deep transverse fibres of the pons into various bundles, whilst in the distal levels they collect gradually into one compact bundle.

Above the pyramidal tracts in the *tegmental region*, we meet the *interolivary layer*, which contains a great number of the sensory conduction tracts, chiefly those serving for the conduction of the deep sensibility (sense of position, etc.) and perhaps for the tactile sense. The change in their position and form can be seen in the illustrations. It cannot be doubted that the transverse field lying above and external to the lemniscus, the formatio reticularis, also contains some of the sensory conduction tracts; these are the spinothalamic and spinotectal as well as the bulbothalamic fibres, which pass through the ventro-lateral dorsal region of the pons, and probably serve for the conduction of pain and temperature stimuli. These tracts for the conduction of pain and temperature, which in the distal portions of the brain stem are separated from the lemniscus, seem to come gradually closer to it in the proximal portions.

The dorso-medial bundle of the posterior longitudinal fasciculus lies below the floor of the fourth ventricle and retains its position throughout the whole pons. Its physiological importance has been discussed on pp. 656 and pp. 698 *et seq.* The *spinal trigeminal root* can be followed entirely through the pons. It lies in the lateral zone. A lesion capable of destroying the roots on both sides would have to be a very extensive one. The so-called secondary or central (nucleo-cortical, quinto-thalamic) trigeminal tract passes, according to Hösel, Wallenberg, Kohnstamm, etc., first through the dorso-medial—below the abducens nucleus—and, nearer the brain, through the dorso-lateral dorsal region of the pons, split into two bundles, whilst Spitzer, along with Held and Kölliker, trace its course through the central part of the dorsal region of the pons, above the median fillet, and eventually in association with it (see p. 643).

The *nuclei of the auditory nerve*, as well as *Deiters' nucleus* or the *nucleus of the vestibular nerve* are situated in the inferior segments of the pons, below the floor of the fourth ventricle, and the *facial nucleus* lies deeply in its lateral part. Higher up we find the *abducens nucleus*, and the abducens root, which generally cuts through the sensory and the motor conduction tracts. Further outwards we come to the area in which are found, to the side in the angle below the floor of the fourth ventricle, the *motor* and *sensory trigeminal nucleus*. The fourth ventricle is now covered in by the velum medullare, whilst the lateral wall is formed by the *superior cerebellar peduncle*. At this level we meet the *trochlear root*, and just above it the trochlear nucleus.

The fourth ventricle now passes into the aqueduct of Sylvius, the superior cerebellar peduncle being pushed inwards and downwards, and the posterior corpora quadrigemina surmounting the aqueduct. We may regard this region as the upper limit of the pons.

Diseases of the pons cannot for the most part be distinguished from those of the medulla oblongata, yet it seems advisable to discuss separately the *symptoms of the focal diseases* which develop in the pons.

It is obvious that the symptoms will essentially depend upon the *extent* and *position* of the focus. Small pontine foci may cause no symptoms of any kind, especially if they are situated in the grey pontine nuclei

or in the transverse fibrous layer. A focus which more or less completely destroys the *pyramidal tract* of one side, produces *hemiplegia*, and if it affects the pyramidal tract in the proximal segments, this hemiplegia is of the ordinary type. But if the lesion is situated not far above the facial nucleus, there is no facial paralysis, since the central facial tract which joins the pyramidal has already crossed the middle line in order to reach the nucleus of the opposite side.

If the focus is so extensive as to involve the neighbouring facial nucleus or the roots, it produces *alternate hemiplegia* (Millard-Gubler type). The facial paralysis is (or may be) in that case *degenerative*, if the nucleus itself or the root emerging from it be affected; it is *simple* and not atrophic if the lesion involves the central facial tract which passes into the nucleus from the other side.

Paralysis of the whole facial, without diminution, but on the contrary exaggeration of the electrical excitability and with increase of the mechanical excitability, seems from my experience to be evidence of an affection of the supra-nuclear facial directly before its entrance into the nucleus. But I give this opinion with reserve.[1]

It is easy to understand that the *abducens* may be slightly affected along with the pyramidal tract. We then have an alternate hemiplegia of the sixth nerve and the other side of the body. Lesions in its neighbourhood (see p. 698) may also give rise to associated paralysis of conjugate deviation towards the side of the lesion, facial paralysis on the same and paralysis of the extremities on the opposite side. Grasset terms this the *Foville type* of alternate hemiplegia.

Raymond-Cestan (*Gaz. des hôp.*, 1903) describe an allied type, in which unilateral focal disease of the proximal area of the pons produces paralysis of conjugate deviation towards one side, hemiparesis with tremor or inco-ordination, and disturbances of the sense of position in the opposite side of the body.

Any of the other bulbar cranial nerves may, of course, be involved along with the opposite side of the body, and we can in this way construct a whole number of types of alternate hemiplegia, such as are contained in the older writings, in particular, upon pontine tumours, and have again been revived in the more recent French literature. See also Varet, *Thèse de Paris*, 1905.

The pons also contains fibres which pass from the cerebrum to other nerve nuclei in the medulla oblongata. Pontine lesions may therefore impair the movements of the *tongue*, *palate*, *larynx*, etc. These disturbances are most strikingly evident when the lesion is an extensive one, implicating *both sides of the pons*. Such a lesion may produce *bilateral paralysis of the extremities*, and by involving the fibres to the muscles of the lips, tongue, palate, and larynx on both sides, may give rise to symptoms of *acute bulbar paralysis* (*q. v.*). Apparently the lesion need only extend slightly beyond the middle line in order to injure these supra-nuclear tracts of the motor cranial nerves on both sides.

Bladder disorders, bilateral especially, are frequently present in pontine disease.

Destruction of the *fillet* of one side causes sensory disturbance on the other side of the body. This disturbance is probably never complete,

[1] These data, given in the third edition of this textbook, have since been confirmed by Babinski (*R. n.*, 1905). It is to be regretted that he has not responded to my evidence on this point (*R. n.*, 1906) with the proper recognition of my priority, but has given an explanation which evades this fact.

as some of the sensory tracts follow other paths (see pp. 638 *et seq.*). Bathy-anæsthesia and incomplete tactile hemianæsthesia are the chief symptoms. Further, a lesion which destroys to a large extent the *formatio reticularis*, may give rise to sensory disturbance. The separation of the tracts for the various kinds of sensation cannot be definitely carried out (Moeli and Marinesco [1]). It seems to be very probable, however, from recent experiments that lesions in the lateral part of the tegmentum may, by causing a lesion of the spino-thalamic and bulbo-thalamic fasciculi of fibres, produce hemianalgesia and thermo-anæsthesia.

These tracts for the most part decussate in the spinal cord; only those fibres which arise from the upper cervical region and serve for the cutaneous innervation of the neck and shoulder cross to the other side at the level of the pyramidal decussation (Wallenberg: see also Kutner-Kramer [2]). Kohnstamm [3] thinks that they pass upwards in the ascending antero-lateral tract or Gowers' tract, and that they terminate largely in the grey masses of the formatio reticularis (centrum sensorium), and from there pass to the thalamus as the bulbo-thalamic tract. These fibres are also joined by the nucleo-cortical tracts of the trigeminus, vagus, etc.

It has been definitely proved in numerous instances that diseases of the pons may cause *ataxia*. In cases of a lesion interrupting one of the fillet tracts, motor ataxia has repeatedly been observed in the limbs of the opposite side, whilst lesions of the restiform body or the spino-cerebellar tracts cause hemiataxia of the same side. Lesion of the *superior peduncles of the cerebellum* and the red nuclei probably produces "*cerebellar ataxia.*" As regards the symptoms of motor irritation, of the character of hemichorea, etc., attributed to these tracts, see p. 693.

Unilateral pontine lesions, situated in the neighbourhood of the abducens nucleus or in the posterior longitudinal bundle, may give rise to the symptom of *associated paralysis of the ocular muscles*. The eyeballs cannot be moved towards the side corresponding to the lesion and deviate towards the other side (see p. 638). Stimulation of the corresponding tracts seems capable of producing nystagmus, and possibly also the analogous symptoms of irritation or contracture in the muscles which rotate the head.

The symptoms caused by lesion of the spinal trigeminal root and the trigeminal nucleus do not require special discussion. Recent cases (Eisenlohr, Bregmann, Wallenberg) point to the fact that the distal portions of the root of the first branch, *i.e.* the dorso-frontal, innervate the mucous membrane, especially of the mouth and tongue, etc., but it has been specially proved by Soelder and Schlesinger that the distribution of the sensory disorders is essentially different in affections of the root area in a peripheral lesion. On this question see also the data of Kutner and Kramer, L. R. Müller,[4] and Rossi.[5]

Lesions in the upper portions of the pons which involve the *corpora quadrigemina* give rise to symptoms which correspond with affection of this region, viz., *paralysis of the ocular muscles*, affecting especially the downward and upward movements of the eyes (Wernicke, Nothnagel, Lichtheim, Parinaud, Kornilow, Gruner-Bertolotti), *changes in the pupils*, *e.g.* mydriasis, miosis, sluggish reaction or absence of the light reflex (Eisenlohr, Bach), *disturbances of the equilibrium*, *diminution of the central acuity of vision* (anterior corpora quadrigemina (?), superior

[1] *A. f. P.*, xxiv. [2] *A. f. P.*, Bd. xlii. [3] *Z. f. N.*, Bd. xxxiv.
[4] *A. f. kl. M.*, Bd. lxxxvi. [5] *Journ. f. Psych.*, ix.

peduncle or external geniculate body), and finally to more or less complete *deafness* and *ataxia*.

Monakow states that lesion of a whole anterior corpora quadrigemina only causes slight diminution of the acuity of vision and does not impair the colour sense. Bach also thinks that it is not yet proved that visual disturbance may be caused by isolated destruction of the corpora quadrigemina. The experiments of Ferrier and Turner support this view.

According to Siebenmann, auditory disturbances are caused by lesion of the lateral fillet, or of the posterior superior peduncle. They may be limited to one side but are usually bilateral. Marburg (*W. kl. W.*, 1905), who has carefully studied focal diseases of the corpora quadrigemina, has come to the conclusion that there are hardly any focal symptoms proper to this region, as both the visual and auditory troubles may be neighbouring symptoms, but he goes too far in his limitation of the area, if he also interprets the paralysis of the ocular muscles in this way. Rothmann's interesting experiments (*N. C.*, 1907, "Beitr. z. Anat, und Phys. d. Ohres," 1908) led him to believe that the posterior corpora quadrigemina is related to the function of hearing, but that it is of less importance than the internal geniculate body, the auditory troubles which occur in lesion of the posterior corpora quadrigemina being thus susceptible of recovery.

Some writers, Nothnagel especially, locate in the pons a so-called *convulsion centre*, irritation of which produces convulsions of the epileptic type. The assumption of a pontine convulsion centre has since been abandoned, although it is not denied that general symptoms of motor irritation may be produced from the pons. Bechterew, who confirmed Nothnagel's observations, is of opinion that the convulsions which occur in lesion of a certain area of the pons (vasomotor centres ?) are dependent on the cerebrum. Luce[1] has again lately affirmed that affections of the pons may give rise to epilepsy ; he regards the motor nuclei of the pons as being of an "epileptogenous nature." Clerk reports a case of this kind.

As we see from this description, the symptoms of pontine diseases are very numerous, but in most cases they are so characteristic that they give a definite indication of the site of the lesion.

As regards the *nature of the lesions* which occur in this region, the pons is a *favourite site for foci of softening*, but not so much for hæmorrhages. Of the tumours, glioma and tubercle are found in this region. *Encephalitis*, especially the form which follows infective diseases (influenza), not infrequently affects the pons. Disseminated sclerosis almost always involves the pons. Further, it should be remembered that an aneurism of the basilar artery may by compression of the pons give rise to corresponding symptoms of irritation and paralysis.

The following case may serve as an example of pontine disease :—

F., aged 13, complained some weeks after an attack of influenza of tingling and heaviness in the left side of the face. A few days later weakness of the right arm and leg appeared. These were followed by a feeling of numbness and uncertainty of movement and by diplopia. The speech became indistinct.

On examination some weeks after the onset of the disease, I found *paralysis of the left facial* in all its branches, with *partial R. D.*, hypæsthesia in the *area of the left trigeminus*, and paralysis of the *left abducens*, along with inability to turn both eyeballs towards the left. The hearing- and bone-conduction were affected on the left side. In the *right* side of the body there was *paresis* of moderate severity, with stiffness and exaggeration of the tendon reflexes. Sensibility was diminished in the right arm and leg, and to a slight degree also in the left arm. In addition there was *ataxia* in the right arm, less marked in the leg, and a trace in the left arm. Speech was somewhat *nasal* and indistinct ; *deglutition* was slightly impaired. *Strangury* was also complained of. There was no change in the fundus of the eye. There was some headache, but no vomiting. These symptoms were caused by an encephalitic focus, which occupied practically the left side of the pons, which it permeated in varying extent

[1] *Z. f. N.*, xv.

from the level of the auditory nucleus to that of the abducens nucleus. As lower down it extended beyond the middle line, it implicated the area of the right fillet tract. The pyramids were only involved for a short way.

In the *medulla oblongata* the various tracts and centres are compressed into a very narrow space, so that lesions of comparatively small size may produce marked paralytic symptoms.

The *motor* conduction tracts in this area lie, like pyramids, in close opposition. If the pyramidal tract is injured above the decussation, hemiplegia is present on the opposite side; when they are interrupted on both sides, paraplegia appears in all four extremities. A focus situated in the pyramidal decussation itself may affect the fibres of one extremity before, and those of the other after the decussation, and thus produce the uncommon symptom of *crossed hemiplegia* (Fig. 362). Wallenberg thinks that the pyramidal fibres of the upper extremities decussate higher up than those for the lower (*i.e.* proximally to the latter). The pyramids probably give off fibres at every level for the motor nerves of the medulla oblongata, which cross the middle line in the raphe. Many other writers think that these tracts become separated from the pyramidal tract proper, even as high as the crus cerebri (see p. 635). According to the investigations of R. Sand, these cortico-bulbar pyramidal fibres leave the main tract only in the pons and form obliquely ascending bundles which penetrate the fillet. The *sensory* tracts are contained partly in the fillet or inter-olivary layer, partly in the formatio reticularis. We have already pointed out the uncertainty which prevails regarding this region. Clinical cases (Senator, Wernicke, Goldscheider, Reinhold, Oordt, Oppenheim, Starr, Marburg-Breuer, Kohnstamm, Rossolimo, Henschen, etc.) and experimental investigations (Bogatschow) make it probable that the tract for deep sensibility (sense of position, etc.) undergoes decussation first in the medulla oblongata, and runs along with the internal arcuate fibres into the fillet of the other side, and in the inter-olivary layer, near the raphe, and that these fibres are therefore contained in the posterior column and fillet. Their lesion would thus cause hemiataxia of the opposite side, whilst lesions in the lower portion of the medulla oblongata might produce ataxia of the same side. Homolateral ataxia may also arise from lesion of the restiform body and the lateral cerebellar tract. According to Mann, Babinski, Nageotte, etc., interruption of one of the tracts going from the restiform body through the cerebellum to the superior cerebellar peduncle may give rise to the symptoms of hemiataxia (or hemiasynergy). This ataxia is not of a sensory character. Porot (*R. n.*, 1906) also saw hemiataxia and hemiasynergy in the case of a lesion which passed right through the superior cerebellar peduncle. Lesion of these parts may also cause cerebellar ataxia (with nystagmus and vertigo?). The tracts for the senses of pain and temperature decussate in the lower portion of the medulla oblongata and in the formatio reticularis (their ventro-lateral segment), and are probably contained in the spino-thalamic and spino-tectal fibres (Edinger, Wallenberg), or in a part of the ventral spino-cerebellar (Gowers) and bulbo-thalamic fasciculi (Kohnstamm). Thus in a case of diabetes, I have seen a fit followed by hemiataxia and bathyanæsthesia on one side and thermo-anæsthesia and analgesia on the other. Cases of this kind, some of them with post-mortem reports, have been published by Wallenberg (*Z. f. N.*, xxvii.), Hun, Starr, Breuer-Marburg, Kohnstamm, Rossolimo (*Z. f. N.*, xxiii.) Henschen (*N. C.*, 1906), E. Müller (*Z. f. N.*, xxxi.), L. R. Müller (*A. f. kl. M.*, Bd. lxxxvi.), and Kutner-Kramer (*A. f. P.*, Bd. xlii.). It is not certain whether the fibres for tactile sensation also run in the formatio reticularis (?) or in the inter-olivary layer, but they seem to pass medially to those for the senses of pain and temperature, and in a great proportion along to the posterior column and fillet. We need not here discuss

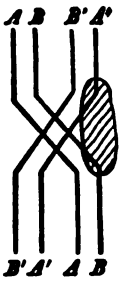


FIG. 363.—Diagrammatic representation of crossed hemiplegia. *A*, right-sided brachial tract; *B*, right-sided crural tract; *A' B'*, corresponding left-sided tracts. The shaded portion shows the injured left-sided brachial and right-sided crural tracts.

another view held by Babinski (*R. n.*, 1906). Much uncertainty still prevails. We lack well-founded experimental evidence as to the function of the olives and the symptoms produced by their lesion. A centre for deglutition has been localised in the olive (and accessory olivary body) (Schroeder van der Kolk, Kesteven, Moeser), but this view lacks confirmation. Bechterew regards the olive as a centre for co-ordination, but its destruction does not always cause disturbance of equilibrium.

It is very remarkable that *some of the nerve nuclei lie close together* in the oblongata, so that a lesion the size of a pea may give rise to bilateral paralysis of these nerves. This is specially the case as regards the *nuclei of the twelfth cranial nerve.*

The structures which form the sensory portion of the trigeminus include the spinal sensory trigeminal root, which lies so near a portion of the great sensory tract that a comparatively small lesion may produce a *crossed or alternate hemianæsthesia, i.e.* anæsthesia in the corresponding fifth nerve and in the opposite side of the body. The anæsthesia may be limited to a part of the trigeminal area (see above and p. 709). The distribution of the fibres and their relation to the conduction of the various kinds of sensation appear to correspond to those of the posterior spinal roots (Gehuchten, Kohnstamm, Rossi, *Journ. f. P.*, ix.).

As a central conduction tract arises from the nucleus of the sensory trigeminus and passes mostly or entirely to the opposite side, a lesion situated in the oblongata or in the pons may produce complete hemianæsthesia or anæsthesia of one side of the trunk and extremities, and of both sides of the face. According to Wallenberg this central trigeminal tract lies in the dorso-medial portion of the medulla oblongata, not far from the hypoglossal nucleus. In one case under my observation a small focus in the oblongata had given rise to *crossed hemihyperæsthesia*, with pain of a corresponding distribution. In another of my cases a focus in the right medulla oblongata had produced a right palato-laryngeal paralysis, singultus, hypæsthesia of the right side of the face, and hemianalgesia and thermo-anæsthesia of the left side of the body. The patient complained of hypersensitiveness of the right side of the body, but the only manifestation of this was that as a merchant he could no longer with the right hand judge cloth stuffs by touch, as the attempt to do so gave rise to an unbearable sensation. Wallenberg also mentions a homolateral hyperæsthesia occurring sometimes in the Brown-Séquard type of bulbar dissociated anæsthesia.

Further, localised lesions of the medulla oblongata often produce the clinical condition of alternate paralysis. Thus unilateral lesions of the eighth to the twelfth cranial nerves may cause paralysis on the same side, associated with hemiplegia of the opposite side of the body. This may also be combined with alternate hemianæsthesia.

Lesion of the vestibular nerve and of Deiters' nucleus gives rise to disturbances of the equilibrium (and vertigo), as shown by the cases of Egger, Wallenberg, Bonnier (*Presse méd.*, 1903), Raymond-Egger (*R. n.*, 1905), and others, but in some of these the conclusions were derived from clinical observation without pathological investigation. A fall towards the side of the lesion has been attributed to lesion of the vestibular nerve, of Deiters' nucleus, or of the descending fibres arising from it or from the cerebellum, but this is not a constant symptom (Wallenberg). Although there can be no doubt as to the relations of this nucleus or of a part of the nuclear group which is formed from the Deiters', vestibular, angular, and other nuclei, to those of the ocular nerves, yet we do not definitely know in how far its lesion may disturb the mechanism of the ocular movements (see pp. 699 *et seq.*). In any case symptoms of irritation (nystagmus), and probably also transient diplopia, may originate from this site. Among the important experiments on animals bearing on this question we would specially refer to that of Winkler ("Internat. Kongress," Amsterdam, 1907), who discusses in detail the course of the motor tracts associated with the eighth nerve.

The medulla oblongata contains in addition a number of *centres*, some of which have a reflex, others an automatic action. These are in part identical with the nerve nuclei which lie on the floor of the fourth ventricle, but their relations are by no means all explained. Flourens localises the site of the respiratory centre behind the point of emergence of the vagus, on both sides of the posterior apex of the fourth ventricle. It is well known that Semon and Horsley found a centre in this region of the fourth ventricle, unilateral stimulation of which produced contraction of the adductors of the vocal cords on both sides, and not far from it another centre from which the abductors could be contracted. These centres have also been found by Dubois-Reymond and Katzenstein. A few cases have, however, been reported in which lesion of this region gave rise to practically no respiratory troubles. Misslawski described a nucleus in the inner zone of the formatio reticularis, the median side of the root of the twelfth, which he regarded as related to

respiration. Gad and Marinesco and also Kohnstamm localise the main portion of the respiratory centre in the grey formatio reticularis. The latter assumes it to be a nucleus of large cells. He regards the respiratory centre as being intimately connected with the sensory nuclei of the vagus and trigeminus and with the tract which serves for the conduction of temperature, especially of cold stimuli. *Respiratory centres* have also been thought to exist in the optic thalamus, on the floor of the third ventricle, and in the posterior corpora quadrigemina. It is a notable fact that respiratory movements have been observed in anencephalus fœtuses (Leonowa), and that the spinal cord may, therefore, to a certain extent regulate this function.

In the neighbourhood of the respiratory centre lies the *cardio-inhibitory centre*. Their independence of each other has been shown by cases of paralysis of the respiratory centres, in which the heart continued to beat for hours if artificial respiration were carried out. Below the floor of the fourth ventricle lies the centre for *deglutition*, which is stimulated from the sensory nerves of the palate and pharynx. The nucleus ambiguus probably represents this centre (see p. 666). Even unilateral lesion of this centre is said to cause disturbance of deglutition (Oordt, Schlesinger, Kohnstamm). The majority of recent writers (Grabower, Wallenberg, Breuer-Marburg, Kohnstamm) regard the nucleus ambiguus as being mainly the motor nucleus for the muscles of the pharynx. These are said to be innervated from the caudal part, and the muscles for deglutition from the proximal part of this nucleus. The attempt of Marinesco-Parhon (*Journ. de Neurol.*, 1907) to differentiate these exactly should be taken into consideration. It is only the part of the act of swallowing that takes place within the mouth that is under voluntary control. As soon as the food enters the pharynx, the swallowing movement becomes a purely reflex one. It has been specially shown by observations on hemicephalics (Kehrer and Hoffmann, Sternberg, etc.), that the sucking reflex and some other phylogenetically old reflexes may originate in the medulla oblongata. The muscles of the lips, tongue, palate, and larynx which control *articulation* and *phonation* are innervated from the corresponding nerve nuclei of the medulla oblongata and pons, which at the same time form the trophic centres of these muscles.

The medulla oblongata also contains, in the neighbourhood of the respiratory centre, a centre for *vomiting*. Kohnstamm has defined centres for the secretion of saliva. He found an upper nucleus of large cells in the region between the facial and trigeminus, dorsal from the facial nucleus, and a lower one between the olive and the nucleus ambiguus. From the former the root bundle is said to pass into the pars intermedia, and by way of the chorda tympani, into the submaxillary gland, whilst the parotid is related to the lower nucleus.

It is well known that *diabetes mellitus* may be produced in animals by injury of the lower part of the fourth ventricle (Claude Bernard). The vasomotor centres are thereby affected. Further experiments have shown that glycosuria may be produced from very various sites of the central nervous system, but very specially from the medulla oblongata (Chauveau and Kaufmann), and that a number of different factors are concerned in the production of this symptom. Pathology shows that diseases of the medulla oblongata sometimes give rise to *melituria*, but this symptom is of very uncertain value, as the conditions under which it occurs have not yet been sufficiently investigated. *Polyuria and albuminuria* have been noted in a few cases of disease of this part of the brain.

We know little for certain as to the localisation of the *vasomotor centres* in the medulla oblongata. The lower *central nuclei* have been assumed to represent these (Bechterew), but so also have other parts. Reinhold (*Z. f. N.*, x.) has thoroughly studied this question and has defined median areas of the subependymary grey matter of the ventricle of comparatively large extent as vasomotor centres. Cassirer ("Die vasomot. troph. Neurosen," Berlin, 1901) has disputed the validity of these observations. Kohnstamm regards the posterior vagus nucleus, which he terms the nucleus sympathicus (or visceral nucleus) of the medulla oblongata, as a vasomotor centre (see p. 497). He also ascribes vasodilator functions to his nucleus salivatorius.

According to Hoffmann, Breuer-Marburg, and others, the pons and medulla oblongata are traversed by a conduction tract which contains oculo-pupillary fibres. Lesion of this tract causes contraction of the pupil and palpebral aperture of the same side. The tract arises from the cerebrum, undergoes decussation before its entrance into the pons, passes through the dorso-medial region of the formatio reticularis, and reaches the area in the spinal cord described as the cilio-spinal centre. E. Müller (*Z. f. N.*, xxxi.) thinks the tract arises in the cervical cord and ascends thence to the brain stem. Focal diseases of the pons and oblongata may, therefore, as the cases of Babinski, Nageotte, Rossolimo, Cestan-Chenais, Hoffmann (*A. f. kl. M.*, Bd. lxxiii.), Cauzard and Laignel-Lavastine (*R. n.*, 1905), L. R. Müller, and others show, produce the symptoms of

" sympathetic opthalmoplegia " (a term used by Breuer and Marburg, but not well chosen). I have occasionally observed this symptom, but in one case in which an alternate hemianæsthesia of the left side of the face and the right side of the body, associated with cerebellar ataxia and falling towards the left, developed suddenly in an old man, the contracted pupils were also insensitive to light, and further examination showed that the lesion had occurred in an individual already suffering from latent tabes.

There are a number of diseases of the central nervous system which involve the medulla oblongata, although it is not the chief seat of the disease. These include *tabes dorsalis, disseminated sclerosis*, and *gliosis*. It has also been found to be involved in a few rare cases of combined posterior and lateral column disease (*q.v.*) (Mayer, Henneberg). Myelitis of the upper cervical cord may also extend into the medulla oblongata, and *progressive muscular atrophy* may spread into it in a systematic way. This portion of the central nervous system is almost always involved in *amyotrophic lateral sclerosis* (*q.v.*).

Let us now pass over those processes which are considered at other chapters, and consider the diseases which arise in the medulla oblongata itself or spread into it from the neighbourhood.

Progressive Bulbar Paralysis[1]

(PROGRESSIVE GLOSSO-PHARYNGO-LABIAL PARALYSIS)

This is a rare disease. It mainly affects people in advanced life, in the fifth and sixth decades. It very rarely appears before the age of 50. It has been thought to occur in childhood in a few cases which were not confirmed by post-mortem examination. This infantile form, however, requires special discussion.

The *causes* are unknown. Chill, emotion, trauma, and specially exhaustion of the muscles of the tongue, lips, and palate, have been held responsible. In one case (Hoffmann) there was chronic lead-poisoning. It is doubtful whether hereditary and congenital tendency plays a part, but there is much to be said for the view that a congenital inferiority of the corresponding nervous structures causes them to fail and to lose their power of function prematurely.

Symptoms.—Difficulty of *speech*, of *deglutition, mastication, and phonation*, developing slowly and produced by a *progressive symmetrical paralysis and atrophy of the muscles of the lips, tongue, palate, pharynx, larynx, and jaws*, shows the nature of the disease.

The speech disturbance is as a rule the earliest symptom. The patient notices that speaking fatigues him, that he becomes exhausted by a long conversation, and that he cannot articulate his words so distinctly as formerly. Those around him also notice this. The linguals, d, t, l, r, n, s, sch, i, etc., are the first to cause difficulty ; they are pronounced indistinctly, and as if they were slurred. Later the labials p, v, f, m, o, and u, especially those which require the lips to be firmly closed, are also involved. At the same time or later, the speech becomes

[1] Literature: Duchenne, " Paralysie muscul. progressive de la langue," etc., *Arch. gén de Méd.*, 1860; Wachsmuth, " Über progressive Bulbärparalyse," etc., Dorpat, 1864; Charcot, *Arch. de Phys.*, 1870; Leyden, *A. f. P.*, ii. and iii.; Kussmaul, " Über die fortschr. Bulb.," etc., *Volkm. Samml. klin. Vortr.*, 1873; Leyden, *A. f. P.*, viii.; Eisenlohr, *Z. f. kl. M.*, i. Further bibliography in Gräfe-Saemisch's " Handbuch," 2nd ed. T. 2, Bd. xi.; in Cassirer, " Die prog. Bulb.," " Handbuch der Path. Anat. d. Nerv.," Bd. i., 1904; see also Bruns, Eulenburg's Realenzyklopädie.

nasal, part of the expiratory current of air passing through the nose. B and p sound like mb, mp. The bulbar speech affection—*dysarthria*—can no longer fail to be recognised. The words are pronounced indistinctly, thickly, and more or less nasally; the patient speaks as if he had a pebble in his mouth. This is less marked in a short than in a longer conversation, as fatigue intensifies the trouble.

In the first stage, or after the dysarthria has been present for some months, *deglutition* also becomes affected. The patient finds it troublesome to pass the food with his tongue from the cavity of the mouth into the pharynx and thence to the œsophagus; fluids regurgitate through the nose or pass into the entrance to the larynx and bring on a fit of coughing. Finally, the dysphagia develops into complete inability to swallow solid or liquid food.

Mastication is often also affected in the further course of the illness. *Phonation* and *respiration* are eventually involved. The voice is weak, monotonous, and without modulation; it is deep-set, and hoarseness, amounting to aphonia, may appear. Coughing may become weak and toneless. Towards the end of life the respiration usually becomes difficult and dyspnœic, and severe attacks of suffocation may ensue.

The functional troubles here described are due to *paralysis* and *degeneration of the muscles*. The tongue is usually first affected. Its larger movements are not necessarily implicated, even although the articulation of the linguals has already become markedly impaired. Gradually, however, the weakness becomes evident when the movements are tested. The tongue is then protruded incompletely and with difficulty; it is soon drawn back again, and usually cannot be moved to the side. The weakness of the lip muscles is shown by the feeble way in which the lips are closed, and by the inability to whistle or point the lips. The soft palate is depressed, and on phonation is raised but slightly, or, later, not at all. As a rule the palatal and laryngeal reflexes are absent. The paralysis subsequently extends to the muscles of deglutition.

Laryngoscopic examination as a rule at first reveals normal conditions, but later the paresis of the adductors may be recognised by defective closure of the rima glottidis in phonation. Weakness of the muscles of mastication is shown by the feeble way in which the jaws are shut, and by the inability to move the lower jaws laterally.

The muscles which originate in and are inserted into the hyoid bone are often involved in the paresis, as Collier (*R. of N.*, 1903) has shown. He is of opinion that loss of function of these muscles plays a part in the dysphagia and dysphonia.

The glosso-pharyngo-labial paralysis is of a *degenerative* nature. The atrophy does not as a rule appear early, and it frequently does not keep pace with the paralysis. The functional disturbances tend to precede for a considerable time the visible atrophy of the muscles. This is first apparent in the tongue, which becomes flaccid, feels soft and spongy, and shows a marked fibrillary tremor. It looks as if thousands of small muscle fibrils were in constant motion. Should the atrophy develop further, furrows and hollows form in the tongue (Fig. 364), which eventually becomes greatly reduced in size, although it may be histologically altered, even if it remains of normal size. The muscles of the lips are usually involved later, but not to such a marked degree. The

lips become thin, and no longer feel as if they contained muscle. The atrophy of the masseter muscles is seldom very pronounced, but they often show a fibrillary tremor at an early stage.

The degeneration of the muscles is revealed by changes in the electrical excitability, usually by *partial reaction of degeneration*, but this may for a long time be masked by the normal reaction of the muscle fibrils which have remained intact. It usually only becomes apparent in the later stages, and must be carefully looked for.

If the disease is far advanced, the *facial expression* will show how serious the disease has become. The mouth is open, the lower lip droops, the corners of the mouth are usually drawn downwards, and the saliva, whether normal or increased in amount, escapes between the lips. The lower half of the face is rigid, whilst the upper half alone gives expression

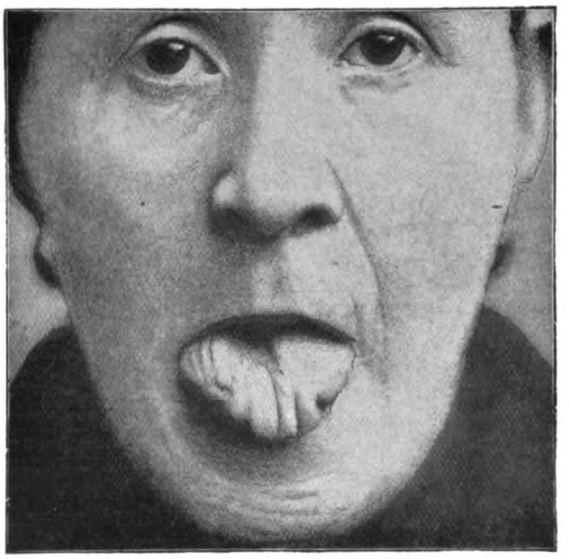


FIG. 364.—Atrophy of the tongue in progressive bulbar paralysis.

to the mental emotions, the eyes and upper facial muscles having retained their normal power of movement. The *upper region of the facial nerve*, viz., the orbicularis palpebralis and frontalis muscles, are very rarely involved in the paralysis, as in a case observed by myself (and Remak), in which there was lagophthalmus, etc. An extension of the paralysis to the ocular muscles—first to the levator palpebraris superior or the abducens—has been noted in rare atypical cases.[1]

The patient weeps readily. In doing so he moves the mouth but slightly, whilst the action of the respiratory muscles assumes a spasmodic character and is sometimes associated with an inspiratory stridor. The tongue lies motionless in the cavity of the mouth ; speech is incomprehensible, as articulation is almost quite impossible ; the power of swallow-

[1] It is stated by Cassirer (*loc. cit.*) that no case of true progressive bulbar paralysis has yet been observed in which there were definite changes of the nuclei of the oculo-motor and trochlear nerves.

ing is lost, the breathing is rapid, and the pulse may be increased to 120 and 140 per minute. The patient is greatly emaciated and is in a helpless condition.

These symptoms are due to paralysis of the motor cranial nerves. In typical cases this paralysis does not extend to the ocular nerves, and it should be specially noted that the sensory cranial nerves are never implicated in the disease. In many cases a *spastic* factor in this paralysis is revealed by the fact that the tendon reflexes are exaggerated in the region of the facial and masseter muscles (see p. 227).

As bulbar paralysis may supervene upon and become combined with progressive muscular atrophy and amyotrophic lateral sclerosis, so also may progressive muscular atrophy and amyotrophic-spastic symptoms in the extremities be superadded to a pre-existing bulbar paralysis, although these naturally do not attain their full development. Thus it is not uncommon for symptoms of glosso-pharyngo-labial paralysis to be accompanied by exaggeration of the tendon reflexes in the extremities and atrophy of some of the muscles of the arm or hand.

Progressive bulbar paralysis is a disease with a slowly advancing course. A deceptive arrest, or less often a remission, may occur. An acute (though not apoplectiform) onset of the speech disturbance has been noted in a few cases, but the subsequent course was steadily progressive. So far as we know, the disease has always a fatal termination, usually in the course of one to three years, but sometimes later. Several cases are known in which death occurred within the first year. The patient succumbs to inanition, bronchitis, broncho-pneumonia, asphyxia, or some intercurrent disease.

Indications for Diagnosis.—Before making a diagnosis of bulbar paralysis, one must ascertain that the speech trouble is not caused by any mechanical impediment (defects in the palate, etc.). The presence of paralysis of the soft palate in no way justifies one in giving this fatal diagnosis, as it may arise from some disease of a comparatively benign character, *e.g.* diphtheria. If, on the other hand, it can be ascertained that the speech disturbance has developed from a slight commencement and has gradually increased, and if symptoms of paralysis are found in several of the muscles concerned in articulation, the diagnosis of progressive bulbar paralysis must be given as the probable, and if atrophy has also appeared, as the only possible one. So long as atrophy has not developed, there is always a possibility of confusion with more benign forms (see bulbar paralysis without pathological change, or myasthenic paralysis). Special care is also necessary in dealing with young patients, as there are curable forms of poliencephalitis which simulate atrophic bulbar paralysis. Although the symptomatology of this condition is usually somewhat different, and its development acute or subacute, yet its onset is occasionally an insidious one.

There is also an *hysterical* and *psychasthenic* form of deglutition paralysis or phagophobia, which is well characterised by its psychogenic origin, but a true dysarthria of this nature, and especially a hysterical paralysis of the soft palate, is so unusual that this diagnosis should only be made from conclusive evidence. In one case in which I was the more easily led to take this view from the fact that the symptom appeared in a singer, at first only periodically and under the influence of excitement and expectation, a true, chronic bulbar paralysis developed after several years. In another case under my care the disease at first appeared to be a speech phobia. There may therefore occasionally be great difficulty in determining the diagnosis.

Acute bulbar paralysis can usually be easily distinguished from pseudo-bulbar paralysis, but some cases of this kind, which will be considered later, may be difficult to diagnose.

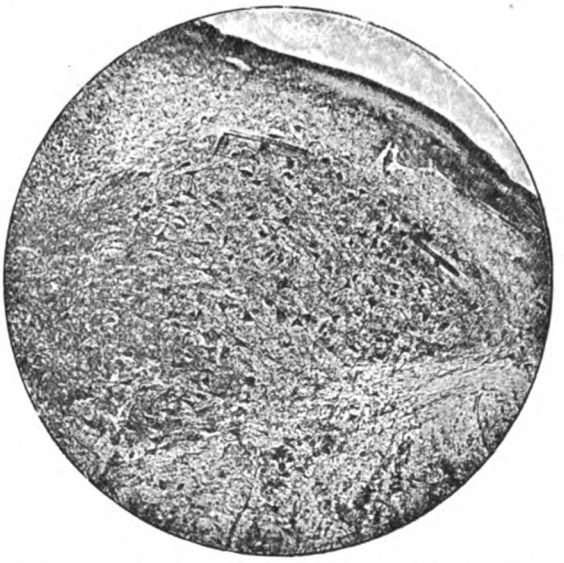


FIG. 365.—Normal hypoglossal nucleus. (Carmin stain.)

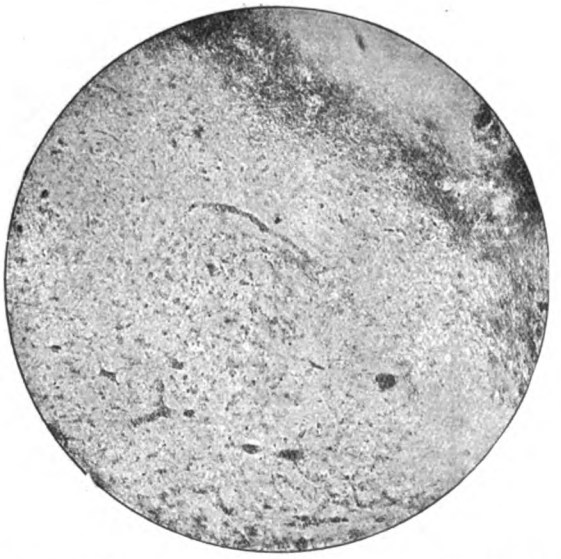


FIG. 366.—(Compare with 365.) Hypoglossal nucleus in progressive atrophic bulbar paralysis. (Carmin stain.)

Tumours of the medulla oblongata also produce symptoms of bulbar paralysis of gradual development, but the paralytic symptoms lack the symmetrical distribution, the selective character, *i.e.* the limitation

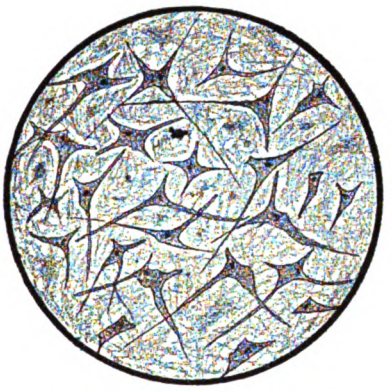


FIG. 367.—From a normal hypoglossal nucleus.

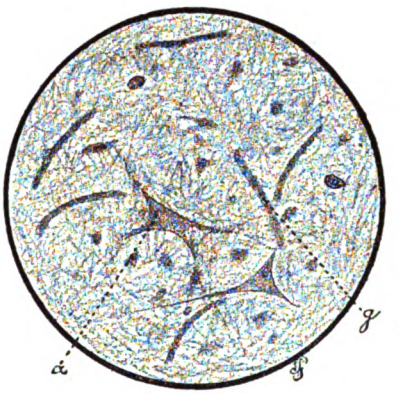


FIG. 368.—(Compare with 367.) From an atrophied hypoglossal nucleus in bulbar paralysis. *g*, vessels.

(Carmin stain.)

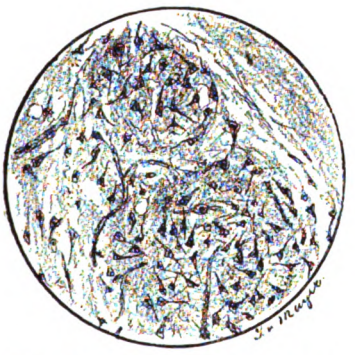


FIG. 369.—Normal facial nucleus.

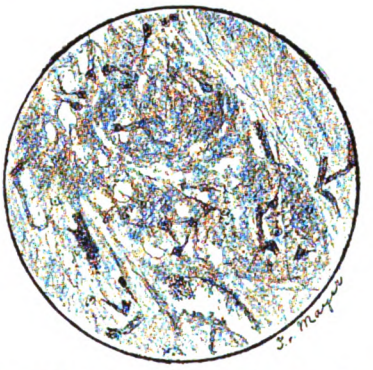


FIG. 370.—Atrophy of the facial nucleus in bulbar paralysis.

(Stained with carmin. Under lower power than in Figs. 367, 368.)

FIG. 371.—Normal medulla oblongata at the level of the hypoglossus; *xii*, hypoglossal nucleus; *w*, hypoglossal roots; *py*, pyramids.

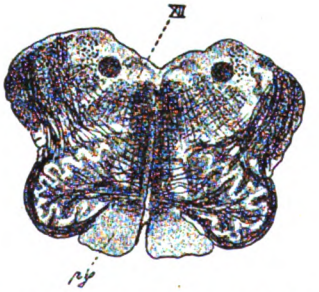


FIG. 372.—Atrophy of the twelfth nucleus and its roots, and of the pyramids, in amyotrophic lateral sclerosis.

(Weigert's stain.)

to the motor nuclei, and are moreover usually accompanied by signs of increased intracranial pressure, which are entirely absent in true bulbar paralysis.

Pathological Anatomy.—The basis of this disease is a lesion of the nuclei of the motor nerves which are situated in the medulla oblongata and the pons, mainly those of the hypoglossal and facial and of the motor glosso-pharyngeal, vagus, and spinal accessory nerves, and sometimes also the nucleus of the motor trigeminus. The nerve-cells which form the most important constituent part of these nuclei, become gradually destroyed; they lose their processes, shrivel up, and finally become completely atrophied (Figs. 366, 368, and 370). The same process affects the intranuclear network of fibres and the intrabulbar and extrabulbar roots. The atrophy of the latter may sometimes be detected by the naked eye. The degeneration naturally also affects the muscles and the intramuscular nerve branches.

The disease may be limited to the grey masses just mentioned, as in some of the cases examined by Charcot, Duchenne, Joffroy, Duval, and Raymond, etc. But the pyramidal tracts are often involved (Fig. 372). The sclerosis of the motor pyramidal tracts may apparently in some cases precede the nuclear atrophy, but it may also be entirely absent, and in cases of this kind, in which the grey matter is alone affected, the course seems generally to be a more rapid one.

The changes characteristic of progressive muscular atrophy and amyotrophic lateral sclerosis (*q.v.*) are often present in the spinal cord.

Treatment.—So far as we know, the disease is incurable. Even as regards prophylaxis there is nothing definite to be said.

As drugs, nitrite of silver, strychnine, iodide of potassium, and arsenic have been recommended. Atropin in doses of 0·0005 ($\frac{1}{1000}$ grain) has been found to control the salivation.

A careful nourishing diet must be given from an early stage. As soon as the danger of choking becomes imminent, artificial feeding by means of an œsophageal tube should be commenced.

Electricity, in the form of direct galvanic treatment of the medulla oblongata, is worth trying in every case. The galvanic current should be passed, under the control of a rheostat, from one mastoid process to the other (with a current of about 2 to 3 milliampères and electrodes of 50 sq. cm.). Galvanic and faradic stimulation should be applied to the muscles of the lips, tongue, and palate, and the galvanic deglutition reflex should be elicited by stimulation of the throat, the anode resting on the nape of the neck, while the cathode is passed from it over the lateral part of the throat.

The experimental use of subcutaneous injections of strychnine may be recommended.

Appendix

The Infantile (Hereditary, Familial) Form of Progressive Bulbar Paralysis

In addition to a few cases, the interpretation of which is uncertain, such as those reported by Berger, J. Hoffmann, Remak, Brown, Naef, and others, a form of progressive bulbar paralysis of childhood has

been specially described by Fazio,[1] Charcot,[2] Londe,[3] and Brissaud-Marie.[4]

The disease is characterised chiefly by its hereditary, familial character, as it has been found to occur in brothers and sisters whose parents are related to each other. Stigmata of heredity and degeneration (prognathism, etc.) have also been found. Another peculiarity is that the paralysis commences in and chiefly affects the region of the *upper facial* (lagophthalmus, etc.), and that it is associated with the bulbar symptom of opthalmoplegia, especially ptosis.

In most cases the paralysis was atrophic and combined with diminution of the electrical excitability and partial reaction of degeneration (generally limited to a small group of muscles). Articulation and deglutition were affected in the typical way. The laryngeal muscles (adductors) were also weakened, whilst the extremities were very rarely involved. Remissions and exacerbations occurred in several cases. Some of the symptoms therefore resembled the myasthenic, others the pseudo-bulbar type (Peritz), although the disease could not be classed with these. Nothing is definitely known as to the *pathological cause*, although it is assumed to be a nuclear disease. In spite of its resemblance to certain forms of myopathy, there can hardly be any suspicion of a primary muscular disease (*cf.* p. 238). On the other hand, especially if we take into consideration Heubner's clinical and pathological investigations, we cannot deny that the affection is related to the form of congenital facial diplegia and paralysis of the ocular muscles (p. 468) due to "infantile nuclear atrophy," as in this case the condition, although resulting from a congenital error of development, only becomes complete during childhood, whereas the latter is a congenital condition. Möbius thus includes along with so-called infantile nuclear atrophy cases in which the symptoms first developed during childhood.

Congenital paralysis of the bulbar nerves has also been described by Berger[5] and Hoppe-Seyler.[6] I shall refer later to another form of infantile glosso-pharyngo-labial paralysis.

Bernhardt (*V. A.*, Bd. cxxvi.) has observed a hereditary disease with symptoms of atrophic bulbar paralysis in adults.

I have once seen bulbar symptoms of uncertain nature associated with a laryngismus stridulus.

The pathological anatomy of the rare cases in which a chronic post-diphtheritic paralysis affected the muscles of the eyes, palate, larynx, and face, is difficult to explain (see W. Harris, *Br.*, 1903), and so is the very interesting case described by Tinel-Siredey (*Bull. et Mém de la Soc. méd. de Paris*, 1906).

The so-called *unilateral bulbar paralysis*, cases of which have been reported by Pel, Erb, Wiener, Geronzi, etc., as a rule resembles the acute forms, and should not be confused with Duchenne's disease (see following chapter).

Acute (Apopleptic) Bulbar Paralysis

The symptoms of glosso-pharyngo-labial paralysis may develop in an acute manner. The pathological changes which underlie this acute form of bulbar paralysis (specially studied by Lichtheim,[7] Leyden,[8] Senator,[9] Eisenlohr,[10] Oppenheim and Siemerling,[11] Wallenberg,[12] and others), vary greatly, but as a rule we have to deal with an affection arising from the *vascular system*, with hæmorrhage, and still more often with *softening* due to *thrombosis*, less frequently to embolism of the *vertebral and basilar arteries*, and their branches.

Hæmorrhage into the substance of the pons and medulla oblongata is on the whole rare, and usually leads to a rapidly fatal termination

[1] *Rif. med.*, 1882.
[2] *Méd. mod.*, 1893.
[3] *Rev. de Méd.*, 1893-94.
[4] *Bull. méd.*, 1893.
[5] *B. k. W.*, 1876, and *V. A.*, Bd. cxxii.
[6] *Z. f. N.*, ii.
[7] *A. f. kl. M.*, 1876.
[8] *A. f. P.*, vii., and *Z. f. kl. M.*, 1882.
[9] *A. f. P.*, xi.
[10] *A. f. P.*, ix. and x.
[11] *Charité-Annalen*, xii.
[12] *Z. f. N.*, xix. and xxvii.; *A. f. P.*, xxxiv.

before the signs of bulbar paralysis have fully developed. In a number of cases, however, single or multiple hæmorrhagic lesions in the pons and medulla oblongata have been shown to be the cause of the illness (Senator,[1] Schulz, Schlesinger,[2] Gee and Tooth,[3] Luce,[4] M. Cohn,[5] Queirolo,[6] Benvenuti,[7] Dana,[8] and others). Cerebral hæmorrhage is the main cause, but it should be specially stated that injuries to the head, especially to the occipital region, may give rise to hæmorrhage in the pons and medulla oblongata.[9]

Foci of softening are much more common in this region. They may be of microscopic size, or so large that they extend from the upper portion of the pons into the medulla oblongata and include a large part of the transverse section. They are very often accompanied by a great number of smaller foci. The softening is almost always the result of occlusion of one of the vessels supplying the bulb. The basilar and vertebral arteries (especially the left) are comparatively often occluded by a thrombus. The thrombosis is caused by an arteritis, due either to atheromatosis or to specific brain disease, which has a tendency to affect the basilar (Fig. 373; *cf.* also Fig. 362) and vertebral arteries. It is not

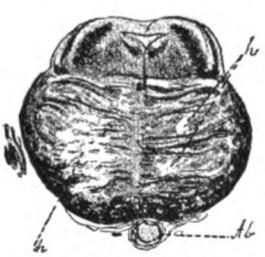


FIG. 373.—Thrombosis of the basilar artery, with foci of softening in the pons in syphilis. (Pal's stain.) *h*, focus.

FIG. 374.—Two foci of softening (*h*) in the pons due to vascular disease.

so usual for embolism from a diseased heart to make its way into the vertebral artery (usually the left). In other cases it is not the arteries themselves, but their branches, *e.g.* the posterior inferior cerebellar artery, which are occluded.

It should, however, be remembered, that in addition to thrombosis of the vessels of the medulla oblongata, arterio-sclerosis gives rise to constriction of these vessels and to disease of their walls, which impairs the circulation. The atheromatous artery is often so greatly dilated and therefore so rigid, that it exercises pressure upon the adjacent tissue of the medulla (Fig. 377). Foci of softening not due to disease of the vessels but to encephalitis or myelitis have been found only in rare cases in the pons and medulla oblongata (Leyden, Etter,[10] Eisenlohr, Oppenheim), and we have some clinical cases which point to this process.

[1] *Charité-Annalen*, xvi.
[2] *Z. f. kl. M.*, Bd. xxxii. Suppl.
[3] *Br.*, 1898.
[4] *Z. f. N.*, xv.
[5] *A. f. P.*, xxxiv.
[6] *Gaz. hebd. de méd.*, 1900.
[7] *Annal. di Nevrol.*, 1901.
[8] *Med. Rec.*, 1903.
[9] In gliosis also acute bulbar paralysis may be suddenly brought on by a hæmorrhage. Schlesinger, who has specially pointed out this fact, has seen symptoms of this kind in a few cases of caisson disease.
[10] "Korresp. f. Schweiz. Ärzte," 1882.

Schlesinger and Hori [1] describe a hæmorrhagic encephalitis of this kind. I have lately seen the disease follow perityphlitis. Bruns reports a case of pontine encephalitis after sausage poisoning. *An acute (and subacute) inferior poliencephalitis*, *i.e.* an acute and subacute inflammation limited to the grey matter of the nuclei of the bulbar nerves, with the symptoms of bulbar paralysis, may also occur (cases of Eisenlohr, Hoppe-Seyler, Geronzi, Oppenheim,[2] Kollarits, Green-Wilson, Taylor, Zappert,[3] etc.; see chapter on encephalitis). The process has been regarded as analogous to acute poliomyelitis, and it has also been observed in childhood and after an infective illness. It may be limited to one side and produce a unilateral bulbar paralysis.

A few cases (Eisenlohr, Schlesinger, Lorenz, Dogliotti, and Cassirer [4]; *cf.* Fig. 325) have proved that the symptoms of bulbar paralysis may be caused by the extremely rare condition of *abscess* of the medulla oblongata. Bulbar neuritis will be considered later.

In describing the symptoms we shall consider only the typical form of acute bulbar paralysis, *i.e.* the form due to softening (and hæmorrhage).

Prodromata in the form of symptoms which are directly caused by the vascular disease, *e.g.* pressure in the head, pain in the occiput and neck, vertigo, insomnia, buzzing in the ears, and swimming of objects before the eyes sometimes occur. The *paralysis* then comes on *with one sudden stroke*. An attack of vertigo, or much less often an apoplectic fit, with complete loss of consciousness, is the first symptom. The vertigo may be so severe that the patient falls down, and it may be accompanied by vomiting. General convulsions of an epileptic character occur only in a few cases. This fit is immediately followed by symptoms of fully developed glosso-pharyngo-labial paralysis. It is only in very rare cases that a couple of days elapse before the disease reaches its full height. A subacute onset, lasting for weeks, has only been noted in exceptional cases of encephalitis, or myelitis and poliencephalitis. *Dysarthria*, or even anarthria, and *dysphagia*, or complete inability to swallow, is present from the first. *Lockjaw*, caused by tonic contraction of the masseters, is often one of the first symptoms, and weakness of these muscles develops during the later course or is present from the onset. There may also be masseter clonus. The muscles in the region of the inferior facial are more or less completely paralysed on both sides,[5] but this paralysis is usually *asymmetrical*. There is glossoplegia, paresis or paralysis of the muscles of the palate and larynx, especially of the adductors, which causes hoarseness or aphonia. The auditory nerve is seldom involved.

In the majority of cases the paralysis extends to the muscles of the extremities, and *paraplegia* of the arms and legs, or *hemiplegia*—usually on the side on which the paralysis of the bulbar nerves is least well marked, and therefore in the form of alternate hemiplegia, or *vice versâ*—or paralysis of one arm and both legs, is present from the commencement. Paralysis of the lower, accompanied by a slight degree of paresis in the

[1] *Obersteiner*, iv.

[2] Bibliography in Oppenheim-Cassirer, "Die Encephalitis," 2nd ed., 1907.

[3] *Jahrb. f. P.*, xxii.

[4] *A. f. P.*, xxxvi., with bibliography.

[5] Symptoms of motor irritation in the facial muscles are seldom caused by these acute affections of the pons, but I have once seen a severe convulsive tic accompanied by characteristic symptoms of paralysis in a case of a lesion in the pons due to embolism or encephalitis.

upper, limbs, is not uncommon. I have only once seen the opposite condition. The hemiplegia may pass from one side to the other. The paralysis of the extremities is as a rule associated with rigidity of the muscles and exaggeration of the tendon reflexes. I have found Babinski's sign absent under these conditions in a surprising number of cases.

Respiratory disturbances (dyspnœa, Cheyne-Stokes breathing, etc.) may be present from the onset or may appear later. They usually occur only towards the end of life, but some cases have been described in which Cheyne-Stokes respiration had existed for months, or even for a year and more. *Increase in the pulse rate* and *rise in the body temperature* to 30-40° C. is not unusual, but this increase as a rule is only very marked towards the end in cases which terminate fatally.

The *facial expression* of the patient is characteristically changed, and the immobility of the features is the more striking from its very sudden onset. Although the senses are usually unaffected and the intelligence unimpaired, one might easily assume the patient to be weak-minded, from the way in which he bursts into *tears* or *laughter* on the slightest occasion. These signs of emotion are often of a *spasmodic* character and produce a tonic contraction of the facial and respiratory muscles, which may result in a condition of dyspnœa (see with regard to this point the chapter on pseudobulbar paralysis).

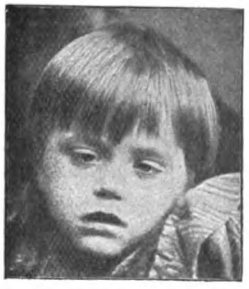


FIG. 375.—(Oppenheim.) Facial expression in paralysis of both facial nerves, abducens nerve, and muscles of mastication caused by diffuse disease of the pons.

We can understand how it is that paralysis of the lip, tongue, and palatal muscles is not at first associated with atrophy, as the latter takes time to develop, but even in the subsequent course it rarely appears, and then only in the parts supplied by a single nerve (*e.g.* in one-half of the tongue or one side of the face). The reason for this is that the lesion does not usually destroy the nucleus and the emerging roots, but injures the *cortico-nuclear* or *supra-nuclear tract*, which passes from the brain to the nucleus on its way through the pons, shortly before its entrance into the grey matter of the nucleus. A portion of the nuclear area may of course be involved, but seldom to such an extent as to give rise to trophic disturbances. Atrophy and involvement of the corresponding reflexes then ensue. Thus in one such case we found atrophy of one side of the tongue, and Rheinhold noted loss of the electrical excitability in the area of the pharyngeal muscles of one side. An exception is formed by the rare cases which were due to an acute or subacute poliencephalitis, an inflammation chiefly of the grey matter, *e.g.* the bulbar form of infantile paralysis. In one case of this kind there was paralysis in all the branches of the facial nerve, with signs of reaction of degeneration; in others there was hemiatrophy of the tongue, which, *e.g.* in cases of so-called unilateral bulbar paralysis, was the most constant symptom, or involvement of several cranial nerves of one side. Cases of a similar kind have been reported by Wiener, Geronzi, and Oppenheim.

These forms of acute and subacute poliencephalitis in children and adults are of rare occurrence, and are distinguished from acute bulbar paralysis by their non-apoplectic development (see also p. 836).

Sensory disturbances may appear in acute bulbar paralysis. The patients not infrequently complain of paræsthesiæ in one side of the body or in one or both arms, and sometimes, as in a case under my observation, of severe *pain* in one side of the body. Hypæsthesia or *anæsthesia* has also been found in the trigeminal region in a few cases (the mucous membrane was not affected), and *alternate or crossed hemianæsthesia* is specially common (Senator, Wallenberg, Oppenheim, Starr, Marburg-Breuer,[1] Hun,[2] Rossolimo,[3] Kohnstamm, Henschen, E. Müller,[4] L. R. Müller,[5] Kutner-Kramer, etc.). It has been pointed out in the introduction to this section that this hemianæsthesia in lesions of the oblongata has often the character of partial sensory paralysis, and may form one of the symptoms of a bulbar type of the Brown-Séquard

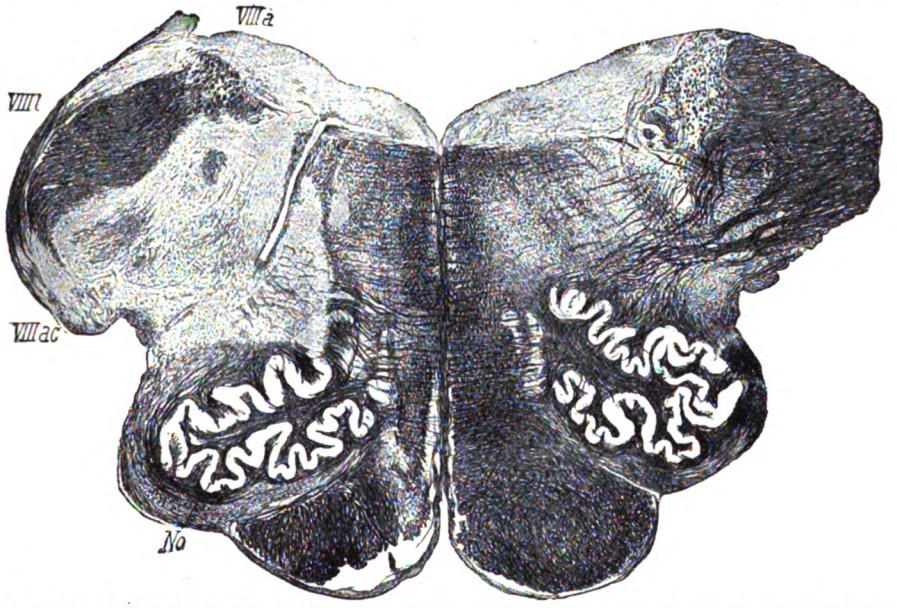


FIG. 376.—Focus of softening in the left half of the medulla oblongata due to thrombosis of the left vertebral artery. (After Breuer and Marburg.) Weigert stain.

syndrome (bathyanæsthesia, with or without ataxia on the same side; analgesia, and thermo-anæsthesia on the opposite side). The conditions under which simple hemianæsthesia or alternate hemianæsthesia, extending to the area of the opposite trigeminus, may appear instead of alternate hemianæsthesia, have also been described there (see also below). A case of Kohnstamm's is of special interest from the fact that the alternate hemianæsthesia implicated only the senses of cold and pain.

E. Müller (*Z. f. N.*, xxi.) describes another variety, in which the hemianæsthesia affected the opposite trigeminal nerve but mainly to its first branch, the cortico-nulcear tract of which decussates lowest down, and was accompanied by a constant objective feeling of warmth. The temperature sense was found to be abolished, or stimuli of indifferent temperatures produced a lukewarm sensation, whilst all the higher temperatures were felt as cooler, and the lower as warmer than on the unaffected side. The sensory disturbance in the trigeminal nerve of the same side may take the form of tactile and deep anæsthesia.

[1] *Obersteiner*, ix.
[2] *N. Y. Med. Journ.*, 1897.
[3] *Z. f. N.*, xxiii.
[4] *Z. f. N.*, xxxi.
[5] *A. f. kl. M.*, Bd. lxxxvi.

In one case of this kind which I examined, thermoparæsthesiæ, perverted sensations of temperature, and a troublesome parageusia (a salt taste) played an important part; moreover, the cold reflex (in Kohnstamm's sense) was absent on the thermo-anæsthetic side, *i.e.* whilst the patient had formerly always been attacked by catarrhal symptoms and sciatic pains when his right leg was uncovered at night, he could now keep this limb uncovered for hours without any ill effect.

We have already referred to the occurrence of bulbar *ataxia*, which has the character of so-called sensory, cerebellar, and motor ataxia, or may show a combination of these types.

Salivation has often been noted. Albuminuria, glycosuria, etc., were rare. Babinski [1] and others have found vasomotor disturbances in focal bulbar disease.

If the lesion extends into the uppermost part of the pons or even into the region of the corpora quadrigemina, *oculomotor* and other symptoms supervene, but it seems that the so-called oculo-pupillary signs may, as noted above, be produced by focal diseases at any level of the pons and medulla oblongata.

It is easy to see that the symptoms must vary according to the size, site, and extension of the lesion. Thus cases have been reported in which the vagus, accessorius, and trigeminus of one side only were affected, others in which the sensory and motor tracts were simultaneously involved, and yet others in which the greater part of the cortico-nuclear fibres and the pyramidal tract were interrupted on both sides, and one facial nucleus was wholly or partially destroyed. In a case observed by Elsholz only a few of the cranial nerves were paralysed, although there was extensive hæmorrhage of the pons; the extremities were not affected. If the softening is confined to one side, it gives rise to the various forms of alternate hemiplegia. In cases in which the *restiform body* was implicated, disturbances of co-ordination, especially swaying and falling towards one side, have been sometimes noted. *Rotatory nystagmus* has also been attributed to it. Lesion of the vestibular and of Deiters' nuclei, and of the fibres originating in them, may also produce vertigo, disturbance of equilibrium and lateropulsion towards the side of the lesion (Wallenberg, Breuer-Marburg, Babinski-Nageotte, Bonnier), but this symptom may under these conditions be absent or disappear (Wallenberg). Affection of the cerebello-olivary fibres or of the cerebello-spinal tracts has also been regarded as the cause of this symptom. Acute focal diseases of the pons may also apparently give rise to transient loss of the knee-jerk (?). In a few cases of this kind there was a lesion of the spino-cerebellar fibre bundles, to which the symptom has been attributed, *e.g.* by Wallenberg. A focus in the pyramidal tract may pass through the fibres for the arm before the decussation, and those for the leg after the decussion, and may thus give rise to symptoms of a *crossed hemiplegia*. It should be remembered that the lesions are usually very irregular in form, so that they may be mainly situated in the dorsal region of one side of the pons, passing only to a very slight extent beyond the middle line into the anterior part of the other side. All this makes it probable that the paralytic symptoms are hardly ever so symmetrically developed as in Duchenne's bulbar paralysis.

The symptoms of acute bulbar paralysis appear in their full intensity and extent in the cases in which the vertebral or basilar artery is occluded

[1] *R. n.*, 1905.

by a thrombus, whilst obstruction of small branches gives rise only to circumscribed symptoms of paralysis.

In spite of Duret's (*Arch. de Physiol.*, 1873) careful studies upon the vascular supply of the bulb, and the investigations of later writers (Wallenberg, Proust, Marburg, and Breuer), we are not as yet able to present a scheme of the symptoms which we might expect to be constantly present in occlusion of the various vessels, as there are great individual differences in the course of the vessels, their relative size, and the blood-supply of the different areas. Nor are the nuclei and roots of the nerves always supplied by the same arteries. As a rule the vertebral artery supplies the nucleus of the hypoglossus and of the accessorius by means of the anterior spinal artery. The vessels for the nuclei of the vago-glosso-pharyngeus and the acusticus arise from the upper part of the vertebral, or from the lower portion of the basilar arteries, but the nucleus ambiguus is supplied from the inferior cerebellar (and therefore from the vertebral) artery. The abducens nucleus receives its branches from the basilar artery. Occlusion of one *vertebral* artery will therefore suspend the function of the ninth to the twelfth cranial nerves and of the spinal root of the fifth on one side; further, where there is only *one* anterior spinal artery, and where it arises from one vertebra only, one portion of it will be more or less affected on both sides. The condition of the extremities also varies, as the pyramidal tract is supplied from the anterior spinal artery, less often from the vertebral, and usually from the small branches which arise before the union of the artery with that of the other side. Thus occlusion of the vertebral artery will produce hemiplegia—on the opposite side if the area above the decussation undergoes softening, on the same side if the softening affects the distal part. If there is only one spinal artery and if this arises only from one vertebral artery (usually the left in this case), its occlusion may cause bilateral hemiplegia. This will also appear if the softening, although unilateral, affects the corresponding pyramidal tracts above and below the decussation. If the pyramidal tract is supplied by the united anterior spinal artery, occlusion of one vertebral does not necessarily cause paralysis of the extremities. If the obstruction is situated in the upper segment of the basilar artery, then, according to Duchenne, the ocular muscles, at least those of the abducens, will be affected. Occlusion of the basilar artery is a much more serious condition, as it is an unpaired vessel, from which the branches for the respiratory centres and tracts arise. Therefore, according to Duret, thrombosis of its lower portion is immediately fatal.

The *posterior inferior cerebellar artery* is not so often the site of embolism or thrombosis, as one may gather from the clinical cases of Wallenberg, Oordt (*Z. f. N.*, viii.), and others. Wallenberg (*A. f. P.*, Bd. xxxiv., and *Z. f. N.*, Bd. xxvii.) has confirmed the diagnosis, which he made from the symptoms, by post-mortem examination, and so has Goldscheider (*D. m. W.*, 1905). A case described by Anton is too complicated to be of use in this matter, and a case of Lamy's (*R. n.*, 1905) is hardly of any clinical value, as the patient died rapidly in a state of coma. The necrosed area lies in the lateral portion of the medulla oblongata and involves the restiform body, the motor vago-glosso-pharyngeal nucleus, the spinal trigeminal root, or the corresponding nucleus, and part of the formatio reticularis, including the internal arcuate fibres (see Fig. 376). Or the olive may be involved, in addition to portions of the cerebellum. The clinical symptoms are: *unilateral* (rarely bilateral) *paralysis of the palate and larynx, trouble in swallowing, anæsthesia* in the corresponding trigeminus, and opposite *hemianæsthesia* (there is only hemiplegia if the pyramidal tract is implicated). The sensory paralysis is usually of the partial order (thermo-anæsthesia and analgesia), whilst bathyanæsthesia on the same side of the body is often found, and much less commonly impairment of tactile sensation. The trigeminus may also be affected on both sides, by lesion of its root and its central tract. The interesting reports and data of F. Müller and Kutner-Kramer, already referred to, should be compared with the above. In a few cases the symptoms also include motor ataxia of the arm, or of the extremities of the same side (Proust, Dumenil, Reinhold, Anton, Breuer-Marburg, Oppenheim, Babinski-Nageotte, L. R. Müller), which is attributed to the restiform body or the lateral cerebellar tract, or to the internal arcuate fibres before their decussation. Vertigo, falling to one side, and nystagmus may also occur. This question has been most minutely studied by Wallenberg and by Marburg and Breuer. They are of opinion that in the motor and sensory tracts of the oblongata, the fibres for the arm and leg run to a certain extent separately, and that Flatau's law of the eccentric position of the long tracts has been proved to hold good with regard to these tracts. Compare the statements of Kutner-Kramer (*A. f. P.*, Bd. xlii.).

Circumscribed foci of softening which develop in the medulla oblongata, may also give rise

to unilateral hypoglossal paralysis (with atrophy) and to crossed hemiplegia, as Oppenheim (*Charité-Annalen*, xii.), Reinhold (*Z. f. N.*, v.), and more especially Revilliod-Goukowsky have shown. In the latter case a lesion diagnosed as lying at the margin of the pyramid and hypoglossal root was found on post-mortem examination at that site.

The combination of anæsthesia in the parts supplied by one trigeminal nerve and paresis of the inferior facial on the opposite side, is described by Schlesinger (*Jahrb. f. P.*, xxii.). The condition which I found in one case of paralysis of the right soft palate and left vocal cord may perhaps be explained by the assumption that a focus had destroyed the superior half of the right nucleus ambiguus and the cortico-nuclear tract going to the inferior half of the left; but this is entirely hypothetical.

It is not always possible to make a precise local diagnosis of lesions in the pons and oblongata, but we may at least say that in the pons they produce symptoms (*e.g.* paralysis of the ocular muscles, total degenerative paralysis of the facial, associated paralysis of conjugate deviation, etc.) which have not been observed in lesions of the medulla oblongata. Oculo-pupillary symptoms (see above) certainly occur in lesions of the oblongata also.

The *course* depends somewhat upon the severity of the symptoms. Thrombosis of the basilar artery has almost always a fatal termination. It is quite conceivable, however, that syphilitic endarteritis of this vessel may cause merely transient complete obstruction, the circulation being restored before death of the tissue has occurred. I think that some of my own cases should be explained in this way.

If we except the most severe cases, in which death occurs within a few days or weeks from broncho-pneumonia and paralysis of the respiration and heart, we may in general term the course of acute bulbar paralysis a *regressive* one. The symptoms reach their highest development at the onset of the disease, whilst at a later stage improvement occurs which may pass into recovery. That such a course is possible, even in the severest cases of this kind, is shown by one which I have reported. A man hitherto healthy, suddenly developed signs of *glosso-pharyngo-labial paralysis*, associated with *paraplegia of all four extremities*. There was also complete *anarthria and dysphagia, lockjaw, rapid pulse, rise of temperature, thirst*, and profuse salivation (especially on electrical stimulation of the facial muscles, etc.). The condition improved in the course of five to six months to such an extent that a left-sided hemiparesis and slightly nasal speech were the only remaining signs of the disease, and the patient now (after seventeen years), but for these symptoms, enjoys good health.

Bruns ("Eulenb. Real-Enzyklopäd.") has observed far-reaching improvement. I have also seen an acute onset and subsequent disappearance of marked bulbar symptoms in the course of diabetes. Thus in one case there were a number of very transient attacks, in which anarthria, dysphagia, and paralysis of conjugate deviation were associated with right-sided spastic hemiplegia. The final attack only lasted for several days, and then entirely passed away (see also p. 823).

The *treatment* of acute bulbar paralysis is practically similar to that of hæmorrhage, softening, etc. It is usually advisable or necessary to use *antisyphilitic* measures. Acute encephalitis of the pons and oblongata, like encephalitis in general, requires antiphlogistic treatment and counter-irritation. In one case large doses of calomel had a wonderful effect.

In such cases the *diet* is very important. Deglutition-paralysis makes it necessary to use a feeding-tube; the danger of broncho-pneumonia must be kept in view. Such patients are apparently unusually susceptible to croupous pneumonia.

The electrical current may be used in the later stages, in the same manner as in the chronic form.

Appendix

Eisenlohr [1] a long time ago described a peculiar group of symptoms, chiefly bulbar, which occurred in young persons in the course of typhoid fever. These consisted of speech disturbance—dysarthria due to paralysis of the muscles of the lips, tongue, and palate—of trouble in swallowing, weakness in the muscles of mastication and in all the extremities, stupor, and in one case optic neuritis. Two of the patients recovered. In these fatal cases *streptococci*—a species related to, but not identical with the citreus—were found, although practically no histological changes were detected. Eisenlohr attributed the symptoms to a *mixed infection*. Seitz [2] has also, in a case of bulbar paralysis, found the brain permeated with bacteria (pneumococci).

We owe a clinical case of this kind to Gascon. A. Henneberg (*Mitt. aus den Hamb. Staatskr.*) has shown that there may be an absence of any recognisable pathological and bacteriological cause for Eisenlohr's syndrome.

Osann (*A. f. P.*, Bd. xlii.) found in lipomatosis, bulbar symptoms which he was able to attribute to degenerative processes in the corresponding nerve nuclei.

Acute Bulbar Neuritis

Multiple neuritis may implicate the nerves arising from the medulla oblongata. The symptoms of glosso-pharyngo-labial paralysis then supervene in the course of a multiple neuritis, and are thereby sufficiently characterised (Kast, Eisenlohr). There is also undoubtedly a neuritis limited to some of the cranial nerves—a "multiple neuritis of cranial nerves"—but in the cases hitherto published bulbar symptoms are not characteristic of the conditions.

In a case of *leucæmia*, Eisenlohr has seen a severe form of bulbar paralysis, with signs of dysarthria, dysphagia, bilateral complete facial paralysis (R.D.), anæsthesia in the area of distribution of the fifth nerve, ageusia, etc. After a duration of four weeks, death resulted from dyspnœa and collapse. Multiple hæmorrhages were found in the sheaths of the bulbar nerves, which also showed a thick, massive infiltration with lymphoid elements. W. Müller has described a similar case, but degenerative processes in the medulla oblongata have also been observed in leukæmia (see p. 189). Comte (*R. n.*, 1906) has described a bulbar paralysis of neuritic origin.

Bulbar Paralysis from Compression

Tumours which grow into the medulla oblongata, or into its neighbourhood (basis, cerebellum), may be accompanied by acute symptoms of bulbar paralysis. Here, however, the development of the whole disease is usually protracted, and the bulbar group of symptoms do not as a rule come on all at once, but in *successive stages*, or symptoms of paralysis which have hitherto been slight, may suddenly become intensified. I have observed an acute onset of bulbar paralysis from

[1] *D. m. W.*, 1893. [2] *V. A.*, Bd. cl.

compression in a case of gummatous meningitis of the posterior cranial fossa. Caries of the upper cervical vertebra and of the occipital bone (Vulpius) may also give rise to this group of symptoms.

Aneurisms of the basilar and vertebral arteries are particularly interesting in this respect. Their symptoms are chiefly caused by the compression which they exert upon the pons and oblongata and the nerves which originate in them (Lebert,[1] Griesinger,[2] Gerhardt, Moeser,[3] Oppenheim - Siemerling,[4] Ladame-Monakow,[5] Massary, and Carton). Aneurisms are much more common in the basilar than in the vertebral artery. In some cases there is a true *aneurism*, possibly the size of a pigeon's egg or larger, and in others a simple *aneurismal dilatation*. Slight degrees of such a dilatation are especially common in the vertebral artery. The formation of aneurisms (see p. 934) is due as a rule to specific or arterio-sclerotic disease of the vessels. An embolism may also give rise to them. Injuries do not play any prominent part in the etiology of aneurisms of this region. The dilated vessel is usually exceedingly tortuous, and presses upon various parts of the pons and medulla oblongata. As a rule there is marked evidence of this pressure: the basal surface of the pons shows compressed hollows, and the olive, pyramids, etc., may be atrophied or softened. In one case which I examined the basal zone of the oblongata was completely softened (Fig. 377). The aneurism may even make its way into the fourth ventricle. In a case examined by Monakow and Ladame, a lesion extending to the cerebellum, pons, and third crus cerebelli had been produced by aneurism of the vertebral artery. I have also in a few cases of slight arteriosclerosis of the vertebral artery[6] succeeded in finding an atrophy of the olives, which could only be recognised by the microscope.

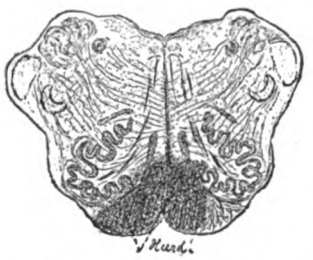


FIG. 377.—Softening of the medulla oblongata from pressure by an aneurismal dilatation of the vertebral artery. (Drawn from a carmin section.)

Aneurisms of the vertebral and basilar arteries may, after prodromal symptoms practically identical with those of cerebral arterio-sclerosis—except that the pain in the occiput and the difficulty in moving the head are specially marked—suddenly produce the bulbar group of symptoms, or the disease may show a gradual development. Attacks, characterised by *anarthria*, *deglutition paralysis*, *dyspnœa*, *rapid pulse and irregular action of the heart*, sometimes also by marked *rise of temperature*, occur repeatedly in the course of the disease. The bulbar symptoms gradually disappear, until a fresh attack brings them back. In the intervals symptoms persist, which may be due to a condition of irritation or paralysis of one or more bulbar nerves, or to a softening of the pons, oblongata, or crus cerebelli (an aneurism of the basilar artery may involve the cerebral peduncle also). Thus rhythmic tremors are observed in the muscles of the face, in the soft palate, and the laryngeal muscles; more frequently there is paralysis of the facial, trigeminus, and vago-acces-

[1] *B. k. W.*, 1866. [2] *A. d. Heilk.*, 1862. [3] *A. f. kl. M.*, Bd. xxxvi.
[4] *Charité-Annalen*, xii. [5] *Nouv. Icon.*, xiii.
[6] This fact, which I described in 1887 (*B. k. W.*, Nr. 34, 1887), has since been confirmed by Marie and Guillain.

sorius, deafness, etc. The latter is peculiarly common (Killian). The *alternate* character of the paralysis found in some well-observed cases is very characteristic. Thus in one of my patients there was paralysis of the vago-accessorius on one side and of the hypoglossus on the other; in another the paralysis involved the soft palate of one side and the facial nerve of the other. The paralysis may be simple or atrophic. The alternate nature of the paralysis of the cranial nerves may be practically explained by the tortuous course of the vessels. There is almost always paralysis of the extremities in the form of general paraplegia, hemiplegia (alternate heimplegia), or paresis, chiefly in the legs. Ataxia, cerebellar ataxia, hemianæsthesia, etc., may be among the symptoms of the disease, *e.g.* in the cases described by Monakow and Ladame. Paralysis of the bladder and rectum is sometimes present.

A valuable sign of aneurism, to which Gerhardt has specially drawn attention, viz., a vascular murmur on the occiput, has hitherto been noted only in a few cases. It is, moreover, certain that such brain murmurs may be caused by tumours of a very vascular nature, by tumours pressing upon a vessel, by hydrocephalus, anæmia, and other conditions (see p. 935). A remarkable symptom has been described by Hallopeau and Giraudau. The patient suffered from respiratory troubles due to aneurism of the basilar artery, and it was found that, as soon as his head, which he kept bent backwards, was moved forwards, his respiration became markedly affected. The breath was arrested in expiration, and only continued when the head was again bent backwards. In another case of aneurism of the posterior communicating artery described by Killian,[1] the head was persistently tilted backwards.

The *prognosis* of these aneurisms is very unfavourable. The disease may last for many months and even for years, but it has almost always a fatal termination, either from softening of the medulla oblongata or from rupture of the aneurism. The symptoms of rupture have already been discussed on p. 935. Recovery is, however, not impossible, especially when the disease has a specific origin.

As syphilis is a very common cause of this disease, anti-syphilitic treatment should be tried in every case. Treatment otherwise resembles that of arterio-sclerosis, of acute bulbar paralysis, and of cerebral aneurisms in general.

Pseudobulbar Paralysis and Cerebro-bulbar Glosso-pharyngo-labial Paralysis

Literature: Charcot, "Arch. de Phys.," 1870; Joffroy, *Gaz. méd. de Paris*, 1872; Jolly, *A. f. P.*, iii.; Lépine, *Rev. mens. de Méd.*, 1877; Eisenlohr, *A. f. P.*, ix. and x.; Kirchhoff, *A. f. P.*, xi.; Wernicke, *Lehrb. d. Gehirnkrankh.*, 1881, and *A. f. P.*, xx.; Ross, *Br.*, 1882; Berger, *Bresl. ärztl. Z.*, 1884; Oppenheim-Siemerling, *B. k. W.*, 1886, *Charité-Annalen*, xii. (in great detail); Leresche, *Thèse de Paris*, 1890; Münzer, *Prag. med. W.*, 1890; Otto, *Z. f. Psych.*, Bd. xlvi.; Senator, *Charité-Annalen*, xvi.; Andereya, "Dissert.," Berlin, 1892; Galavielle, *Thèse de Montpellier*, 1893; Halipré, *Thèse de Paris*, 1894; Jacobsohn, *A. f. P.*, xxvii.; Oppenheim, *Fortschr. d. Med.*, 1895, and *B. k. W.*, 1895; Lépine, *Rev. de Méd.*, 1896; Brissaud, *Rev. scient.*, 1894, and "Leçons sur les malad. nerv.,"1 895; Rose, *Z. f. k. M.*, xxxv.; Perwuschin, "Wratsch," 1900; Comte, "Paralysies pseudobulbaires," Paris, 1900; Roth, *N. C.*, 1901; Urstein, "Dissert.," Berlin, 1900; Polenow, *N. C.*, 1902; Oppenheim, *M. f. P.*, 1903; Hartmann, *Z. f. Heilk.*, 1902; Peritz, *loc. cit.*; Muratoff, *Rev. russ. de Méd.*, 1903; Bruns, "Eulenburgs Realenzyklopädie"; Fürnrohr, *Z. f. N.*, xxvii.; Weisenburg, *Univ. of Penn.*, 1905; H. Müller, *A. f. P.*, Bd. xl.; Charpentier,

[1] *Inaug. Diss.*, Würzberg, 1879.

Rev. de Psych., 1904; Naunyn, *Volkmanns Samml. klin. Vortr.*, 1905; Hoesslin-Selling, *M. m. W.*, 1906; Raymond-Alquier, *R. n.*, 1907.

The symptoms of paralysis of the tongue and lips may also be produced by *diseases of the cerebrum*. The cortical centres of the facial, hypoglossal, motor fifth, vagus, and accessory nerves are connected by conduction tracts with the nuclei in the medulla oblongata and pons. A unilateral lesion of these centres and of the cortico-bulbar conduction tracts is, of course, (with rare exceptions) incapable of producing the symptoms of bulbar paralysis. It may give rise to a unilateral facial and hypoglossal paresis, whilst the masseter, deglutition, and laryngeal muscles are probably only paralysed by bilateral destruction of the cortical centres or conduction tracts.

The muscles for articulation, phonation, mastication, and deglutition have, as already shown on p. 685, a bilateral cortical innervation, so that they are not essentially injured by disease of one hemisphere. On the other hand bilateral destruction of their centres (at the foot of the anterior central convolution), or of their conduction tracts, gives rise to marked functional disturbance, which is the more severe as their cortical centres are situated in a very circumscribed area. In all probability the central ganglia, especially the optic thalamus, also contain centres for these motor actions, which are essentially automatic. Practically all that is required to set these centres into action is an impulse from the cortex, as they are less subject to voluntary control than to the effect of emotional and reflex impulses (see p. 651). Bilateral lesion of these centres or of the tracts which they excite by cortico-fugal or bulbo-centripetal means, or which convey the impulses from them to the deeper centres, also gives rise to *bulbar symptoms*.

These forms of bulbar paralysis, which have their cause and origin, not in an affection of the nuclei of the bulbar nerves, but in a lesion of the *supra-nuclear* tracts or centres, are known as *pseudo-bulbar paralysis*.

Cases of this kind, in which multiple lesions in both hemispheres had produced the symptoms of glosso-pharyngo-labial paralysis, whilst the pons and medulla and the nerves arising from them were found to be intact, were first described by Charcot-Joffroy, Lepine, Barlow, and Jolly. In one of these cases a cerebral form of multiple sclerosis was the cause of the disease; in some others (Piperkoff, Halban, etc.) there were encephalitic foci; in the great majority the pseudo-bulbar paralysis was due to *arterio-sclerosis* or *atheromatosis*, which had produced changes in both hemispheres. The focal disease has occasionally been found to have an embolic origin.

Oppenheim and Siemerling in 1886, on the ground of their investigation of numerous cases of this kind, stated their opinion that pure pseudo-bulbar paralysis was a rare disease, and that focal changes in the pons and medulla oblongata were usually also present, so that as a rule the cases represented a *cerebro-bulbar* form of glosso-pharyngo-labial paralysis. This view was subsequently supported or confirmed by Senator, Otto Jacobsohn, Guizzetti-Ugolotti, Weisenburg, H. Müller, and others. During the last ten years, however, numerous cases of true pseudo-bulbar paralysis have been published (Lépine, Becker, Galavielle, Leresche, Brissaud, Münzer, Oppenheim, Dejerine, Halipré, Fournier, Parhon-Goldstein, Charpentier, Hoesslin-Selling, etc.), and certain definite conclusions have been arrived at from comprehensive studies, amongst which those of Urstein, Goldstein, Peritz, and F. Hartmann should be specially mentioned;

Pathological Anatomy.—The cause of the disease is usually athero-

matosis of the cerebral vessels, with the numerous secondary conditions to which it gives rise, viz., multiple foci of softening, hæmorrhages, apoplectic cysts, local formation of cavities or chronic senile encephalitis in the sense of Marie-Ferrand, etc. Specific arteritis not infrequently produces changes, *i.e.* morbid foci in both hemispheres, which produce the symptoms of pseudo-bulbar paralysis. Multiple embolic, encephalitic, and sclerotic foci have been found to be the cause only in exceptional cases.

The focal lesions very rarely occupy the motor cortical centres, in particular the operculum of Rolando. As a rule they are situated in the *subcortical centrum ovale*—especially corresponding to the posterior portions of the frontal lobe (F. Hartmann)—in the *internal capsule*, and in the *central ganglia*. French writers, Lépine, Brissaud, Dupré-Devaux, Marie, with whom Mingazzini and F. Hartmann to a certain extent agree, attribute a specially important part in the causation of these symptoms to bilateral disease of the lenticular nucleus, assuming that this ganglion contains a centre for the combined movements of deglutition, mastication, etc. Lesion of the ansa lenticularis and of the conduction tract belonging to it would have the same effect. Although it is true that focal diseases are often situated in the putamen of the lenticular nucleus, they may possibly injure one of the cortico-nuclear conduction tracts of the motor cranial nerves in its neighbourhood.

The importance of the optic thalamus as regards involuntary movements (see p. 651) makes it easy to understand why lesion of this ganglion and its tracts should be apt to cause clinical features characteristic of pseudobulbar paralysis.

These subcortical ganglia may not only be to a certain extent stimulated from the higher cortical centres—they may also be *inhibited*. Destruction of these centres or of their tracts to the central ganglia, therefore, suppresses the inhibitory influences and thus leads to an *unrestrained functioning of the lower centres*, which is manifested by symptoms of various kinds.

From this description it is obvious that the clinical picture of pseudobulbar paralysis will vary according to the site, number, and extent of the foci. Moreover we must take into account the fact upon which we have insisted, that atheromatosis may impair the functions of the brain not only by means of the brain disease itself, but in various other ways, *e.g.* by disturbance of the circulation, by pressure from the rigid, tortuous vessels upon the adjacent parts of the brain and cranial nerves, etc. etc. This conception has been recently defended by Naunyn.

Symptomatology.—The symptoms of the disease hardly ever come on in a sudden attack. Rare cases have been described (Magnus, Kirchhof, Bamberger, Polenow, Burr-M'Carthy, Perwuschin, Concetti), in which bulbar symptoms have been attributed to *unilateral* foci in the cerebrum, but even these few cases are not all above criticism. Although it must therefore be admitted that in rare cases and under unusual individual conditions, a *sudden attack* may usher in the symptoms of cerebral glosso-pharyngeal paralysis, yet in the great majority of cases the development of the syndrome of glosso-pharyngo-labial paralysis is associated with two or more apoplectic fits. The disease begins therefore, as a rule, by a successive series of attacks.

These apoplectic fits are not always definitely marked; small foci

may, as shown by the investigations of Marie-Ferrand and ourselves, form so gradually that their symptoms also develop and mature gradually, or in a partly apoplectic, partly chronic manner.

As a rule the bulbar symptoms are associated with bilateral hemiplegia, which, however, is usually incomplete or marked on one side only. The paralysis of the extremities may also be unilateral, and in exceptional cases the cerebral glosso-pharyngo-labial paralysis is a pure one, *i.e.* it is not accompanied by paralysis of the body. But it can almost always be ascertained that transient symptoms, at least of hemiplegia, have been present.

The most prominent symptoms are those which have given the name to the disease, viz., *dysarthria*, *dysphagia*, *dysmasesia* (difficult mastication), and disturbances of *phonation*, *respiration*, and of the movements of facial expression are also common.

When we examine these symptoms in detail, it is at once apparent that the functional disturbance is due chiefly to an impairment of the corresponding voluntary movements, *i.e.* there is paresis or paralysis of the muscles of the lips, tongue, palate, and pharynx, of the masseters, and sometimes also of the muscles of phonation. An important feature of this paralysis is its *non-atrophic* character; the muscles retain their normal size and their electrical excitability, even when the paralysis is of long duration. Another peculiarity is that the muscles which are deprived of voluntary control, may be put into action by *emotional*, *automatic*, and *reflex* means. And finally we should note that in addition to the paresis, spastic symptoms may be present.

Speech is badly articulated and nasal, or it may become an incomprehensible lalling. A kind of stuttering occurs in exceptional cases (Abadie). The patient while swallowing begins to cough, and part of the fluid is regurgitated through the nose, but in pure cases these troubles do not amount to complete aphagia. Mastication is sometimes impaired. Although persistent disturbance of phonation is not frequent (cases of Eisenlohr, Colman, Oppenheim, Hartmann, Münzer, Sendziak [see p. 623]), the abnormalities of respiration and of the movements of expression may prevent the free play of the laryngeal muscles.

In some of my cases the influence of emotion upon the functions of speech and deglutition was so extremely exaggerated that the dysarthria and dysphagia developed under the influence of mental excitement into anarthria, aphonia, and aphagia.

The weakness of the muscles of the lips, tongue, and palate becomes very evident in simple attempts at movement, in the effort to pout the lips, to protrude the tongue, to intone an "a," etc. These may produce abnormal conditions of contraction in the muscles. This spasm is specially marked in the masseter muscles; that is to say there may be true *trismus*.

Other phenomena of motor irritation in the bulbar muscles are much less common. We have described attacks of *grinding the teeth*, and F. Hartmann has observed spasms of the muscles of mastication, which occurred chiefly during night.

It is a fact of the greatest interest that the muscular paralysis is not absolute, that muscles which are no longer under voluntary control can be put into action by emotional, associative, or automatic and reflex

means, and may thus develop an *excessive* activity. Thus it is a common occurrence for the muscles which fail to act in speaking, etc., to take an active part in the movements of expression. Among the most marked symptoms of the disease are *exaggeration* and *modification of the actions of the facial muscles and spasmodic, explosive bursts of weeping and laughing.* In these the muscles of the face which cannot be voluntarily moved show marked and even excessive contraction.

Siemerling and I were the first to point to this sign as a symptom of pseudo-bulbar paralysis and to discuss it in detail ("Mitteilungen über Pseudobulbärparalyse und akute Bulbärparalyse," *Berl. klin. Woch.*, 1886, Nr. 46). In spite of my repeated references to it, this analysis has been neglected by all the later writers, and has been put into the background by the teaching of Bechterew and Brissaud; I shall therefore repeat our statements: "In every case a marked and very prominent morbid symptom occurs, viz., that the patient on attempting to speak bursts into spasmodic weeping and laughing. The facial muscles are contracted to their utmost; the face becomes red or livid; respiration is arrested at the phase of expiration; the pulse becomes small, irregular, or may even stop. The morbid expression is, therefore, shown on the one hand by the exaggerated action of the muscles, which takes the form of tonic spasm of the facial and respiratory muscles, on the other by the fact that the facial movements are elicited with abnormal facility, so that every change in the mind may produce activity in the muscles of the face, in the way just described. It should also be noted that the facial muscles which respond defectively to the will and in articulation, may contract strongly under the influence of emotion, whereby the asymmetry caused by the inequality of the paresis may be adjusted."

We expressed our opinion that these cases were caused by lesion of the centres or by interruption of the tracts which have an inhibitory effect upon the bulbar centres. Explanations of a similar kind, though differing somewhat in detail, have been suggested by Bechterew (*A. f. P.*, xxvi.), Brissaud, and Strümpell. Bechterew localised the centres for the movements of expression in the optic thalamus. These facial movements may be affected in every possible way according as the morbid foci have an irritative or a paralysing effect, and according as they occupy the centres themselves or the tracts which pass to and from them. Brissaud favours the view that emotional movements are conducted through a special tract into the anterior peduncle of the internal capsule, this tract not being identical with the motor conduction path.

The question has more recently been studied by other writers, such as Parhon-Goldstein, Dupré-Devaux, Toulzac, Ferrand, Schaffer (*C. f. N.*, 1903), Deroubaix (*Journ. de Neurol.*, 1906), Wiesner (*W. kl. R.*, 1906), Peigerova (*Casop. lek.*, 1905), Franceschi (*Riv. di Patol.*, x.), and specially by F. Hartmann. This writer essentially confirms our data, describes the rigid, mask-like expression of the face at rest, and the production of the movements of facial expression by many different kinds of stimuli, the "explosive" and "cataleptic" character of these emotional movements, the "*mimische Luxus*," as he calls it, etc. Referring to the investigations of Bikeles, Jacob, Anton, and others, he is inclined to regard the absence of centripetal impulses as a factor in the production of these symptoms. I cannot here discuss whether Sternberg (*W. kl. R.*, 1903) is right in his view that the deeper segments of the brain stem or medulla oblongata contain reflex centres for the expressions of repugnance.

We have also found that in some cases the eyelids could not be actively closed, but that this movement occurred during the spasms of laughing and weeping, that the spasmodically clenched jaws opened whilst the patient was sobbing, etc. In a few cases it seemed to me that an attempt to move the eyeballs to the side was specially apt to bring on spasms of laughter.

This peculiar character of the paralysis is in some cases very strikingly marked in the *ocular muscles.* Whilst voluntary movement of the eyeballs to the side, or upwards and downwards, was difficult, the patient could follow an object with his eyes (Wernicke, Senator, Knies, Oppenheim, Strümpell, Roth, Hartmann), or movement might be produced by a noise. Wernicke was the first to suggest the idea of "pseudo-ophthalmoplegia" (paralysis of the eye-movements from bilateral interruption of the corresponding cortico-nuclear tracts, or bilateral focal disease of the inferior parietal lobe ?) (see p. 699).

In addition to the *respiratory disturbances* associated with the affection of the movements of expression, there may be attacks of *dyspnœa*, *Cheyne-Stokes respiration*, etc. These occur spontaneously or in attempts at movement, after mental excitement, etc. A paroxysm of this kind may last for an hour or more. The pulse may also be rapid and irregular, and the temperature raised, but in view of the multiplicity of the nature and site of the changes caused by the atheromatosis it is difficult to decide whether and to what extent these symptoms should be attributed to pure pseudo-bulbar paralysis. F. Hartmann draws special attention to the dissociation of the movements of articulation and respiration (defective breathing in speaking, swallowing, etc.).

The extremities are usually involved and show simple or bilateral hemiplegia. This is generally incomplete, in the sense that the legs are more markedly affected than the arms (Oppenheim-Siemerling, F. Hartmann). The paralysis of the extremities is spastic in character, as a rule, but this is not always the case. The condition may depend upon whether the motor conduction tract or the central ganglia are implicated, or whether, in addition to the foci which injure the pyramids, there are others so localised that they compensate the hypertonia or counteract the occurrence of the spastic phenomena (Babinski sign, etc.) (see pp. 687, 688).

Atrophy of single muscles in the extremities, which could be attributed to small arterio-sclerotic foci in the spinal cord (see p. 331), was found only in exceptional cases.

The symptoms of hemichorea or athetosis, or bilateral-athetotic movements, and hemichoreic movements in the extremities, have occasionally been observed by ourselves and others.

The *functions of the bladder and rectum* may be impaired or entirely normal. It has been specially shown by Czylharz-Marburg and Homburger that bilateral focal diseases of the central ganglia may cause persistent disturbances of the functions of the bladder and rectum (see pp. 625 and 651). I have also observed undoubted cases of this kind.

As conditions secondary to the vascular process may develop in any part of the brain, it is conceivable that focal symptoms of very different kinds may occur in this disease. Thus aphasia in its different forms is a common symptom. Optic aphasia, mind blindness, central deafness (which in Hartmann's case was caused by bilateral lesion of the internal geniculate body or of the auditory conduction), disturbances of the power of orientation (Hartmann), hemianopia, astereognosis and hemianæsthesia may occur. Thus under these conditions I have occasionally observed the combination of a bilateral homonymous hemianopsia with a contralateral hemiplegia. But it is very remarkable that multiple lesions of the brain almost always lead to *mental impairment*, and that *dementia*, *apathy*, *confusion*, and *conditions of excitement* are almost constant symptoms of the fully developed disease. The marked motor disturbance and the abnormal condition of the facial movements of expression are certainly calculated to simulate mental weakness, but we must recognise the fact that the mental life is unfavourably affected by the disease.

The restlessness of senility (see p. 821) may be superadded to the symptoms described (Naunyn).

Although an affection of the optic nerve does not form part of a

pseudo-bulbar paralysis as we strictly conceive it, yet the nature of the pathological process is such that the changes which develop in the optic nerve in arteriosclerosis, viz., slight degrees of atrophy, and in exceptional cases marked neuritis or atrophy, may occasionally develop here also.

The study of the subcortical, and in particular of the *bulbar reflexes* in these diseases has been greatly advanced of late years. Thus I have shown that in the infantile form of this disease (see the following section and p. 673), bilateral interruption of the motor cortical centres or cortico-nuclear tracts causes exaggeration of certain reflex acts, so that, *e.g.*, stroking of the lips or tongue may elicit a number of rhythmic movements of the lips, jaws, tongue, and pharynx—a true *feed-reflex*.[1] I have also thought that the exaggerated acustico-motor reaction (see p. 851) might be due to the absence of inhibitory impulses. In one of my cases the acustico-motor reflex movement was manifested by the fact that noises produced, in addition to the tonic spasm of the extremities, lateral rotation of the head and eyes. Hartmann has also mentioned the increased effect of noises upon the pulse and respiration. Perrero (*R. n.*, 1902) saw touch on the soft palate produce a reflex movement in the opposite facial. Whether the sucking movement evoked by percussion of the upper lip, described by Toulouse and Vurpas, also occurs in pseudobulbar paralysis, must be proved by further observations. We also require confirmation regarding the "hard palate reflex," described by Lähr and Henneberg (*A. f. P.*, Bd. xxxviii.), *i.e.* a contraction of the muscles of the mouth produced by stroking the hard palate. Henneberg has mentioned its occurrence in pseudobulbar paralysis.

We should also here notice an interesting observation of Jastrowitz and Gutmann (ref. in *D. m. W.*, 1907), who found that a touch on the cornea at the moment of death might provoke the movements of swallowing.

The *diagnosis* of pseudobulbar paralysis is not a difficult matter. The presence of general arterio-sclerosis, the onset of the paralytic symptoms after a single apoplectiform attack or after a succession of seizures, the combination of bulbar symptoms with hemiplegia, diplegia, or paraplegia of a spastic nature, the non-atrophic character of the paralysis, the mental disturbances, etc., all distinguish the disease from progressive bulbar paralysis, even although the spastic paralysis of the legs is found in the form of this disease which is associated with amyotrophic lateral sclerosis. Involvement of the optic, auditory, and sensory trigeminal nerves, aphasia, hemianopsia, etc., are always opposed to the diagnosis of Duchenne's disease.

The differentiation of pseudo-bulbar paralysis from acute, apoplectiform bulbar paralysis is not so easy, and the distinction not so sharp. In the latter the syndrome is as a rule produced by a *single* attack. Moreover the paralytic symptoms simulate those of a diffuse interruption of conduction in the pons or medulla oblongata, and they may, corresponding to the lesion of one or more nerve nuclei, have a degenerative (atrophic) character, although this is rare and slight in extent. In acute bulbar paralysis there are naturally no symptoms arising from the cerebrum. The symptom of the paralysed muscles being called into action through emotion or in an automatic manner, and thus becoming excessively active, is practically confined to pseudobulbar paralysis. This is essentially the case also as regards the exaggeration of the bulbar reflexes, although it must not be forgotten that foci of softening in the

[1] It has been shown experimentally (Oppenheim, Réthi, Horsley-Beevor) that there are sites in the motor region of the cortex where stimulation produces a succession of movements of mastication and deglutition. This has also been demonstrated by Vogt on the brain of the ape. We may assume that both stimulating and inhibiting impulses arise from these sites. We have a few noteworthy data regarding the course of the corresponding conduction tracts (Réthi, Carpenter, Probst, Economico, etc.), but the question requires further study. The rôle played by the substantia nigra in these functions is also as yet uncertain.

proximal parts of the pons give rise to a supranuclear bulbar paralysis, in which the reflexes originating in the medulla oblongata may be exaggerated. Moreover, some of the reflexes in question, *e.g.* the uvular, are normally so inconstant, that they are not of much importance as regards the differential diagnosis.

It is shown by some of our cases, and specially stated by Naunyn, that the clinical picture may be closely allied to that of multiple sclerosis. The disease is distinguished from amyotrophic lateral sclerosis and from the unilateral and bilateral progressive hemiplegia of Spiller (see p. 825) by the slow and steady progress of the latter, and by the purely motor character of these morbid forms.

The distinction between pure pseudobulbar paralysis and the cerebrobulbar form should be made according to the principles already indicated, but it is advisable as a general rule not to make this distinction, and the possibility that the pathological process may have involved the bulb should always be borne in mind.

Amongst the unusual complications of pseudobulbar paralysis, I shall only mention that of tabes, which occurred in a case under my own observation.

The *prognosis* of this condition is unfavourable, but many years may elapse before death ensues, and even the severe paralytic symptoms may show considerable improvement for a certain length of time.

Infantile Form of Pseudobulbar Paralysis

I [1] have described a special form of cerebral bulbar paralysis, and Bouchaud [2] shortly afterwards published a similar case. An article of Binswanger's in the older literature should also be included with these. Clinical cases of this kind have subsequently been reported by König, Brauer, Ganghofner, Collier, Halban, Zahn, Kaufmann, Variot and Roy, Decroly,[3] Schüller,[4] Armand-Delille, and Giry,[5] and especially by Peritz,[6] who collected our material. In these cases the symptoms of glosso-pharyngo-labial paralysis developed along with those of infantile cerebral paralysis, and thus produced an *infantile form of pseudo-bulbar paralysis*. This condition belongs to the category of the diplegias, and is characterised by the association of a bilateral paralysis or paresis (which may be combined with spastic-athetotic symptoms) of the muscles of the lips, tongue, palate, pharynx, and larynx, with the corresponding symptoms of dysarthria, dysphagia, etc. In two of my cases there was trismus, in one absolute dumbness, and in a third aphonia and a phonation spasm. In some of these cases I have found exaggeration of the subcortical, especially of the bulbar reflexes, so that stroking of the lips and tongue produced a number of rhythmic movements of mastication, sucking, and swallowing, a symptom which I have named the "*feed reflex.*" Such movements may also be automatically produced (perhaps by swallowing saliva). Hartmann describes persistent involuntary movements of the jaws. The degenerative atrophy, fibrillary tremor, etc., are naturally not present in the muscles of the head. I have been able to trace the disease to a bilateral disease or malformation (*microgyria*, *porencephaly*)

[1] *N. C.*, 1895, and *B. k. W.*, 1895. [2] *Rev. de Méd.*, 1895. [3] *Journ. de Neurol.*, 1903.
[4] *W. kl. R.*, 1905. [5] *R. n.*, 1907.
[6] "Pseudobulbär- und Bulbärparalysen des Kindesalters," Berlin, 1902; see the bibliography here.

of the lower segment of the central convolutions.[1] Bouchaud and Zahn have subsequently published similar reports. The affection may undoubtedly be acquired after birth from a bilateral focal disease, *e.g.* encephalitis in the inferior central region. The cases described by Halban,[2] Huet and Sicard, Raymond and Lejonne (*R. n.*, 1906), should probably be interpreted in this way. F. Hartmann takes a similar view of one of the cases which he has published.

I have since observed a great number of other cases. Some special features have already been mentioned on p. 850.

Bulbar Paralysis without Pathological Lesion, or Myasthenic Paralysis

(MYASTHENIA GRAVIS PSEUDOPARALYTICA, ASTHENIC BULBAR PARALYSIS, ASTHENIC PARALYSIS)

Literature: Previous to 1901, in H. Oppenheim, "Die myasthenische Paralyse," Berlin, 1901; also, Oppenheim in the "Handbuch d. Path. Anat. d. Nerv.," Berlin, 1904; Wilbrand-Saenger, "Die Neurologie des Auges"; Murri, *Riv. crit. di clin. med.*, 1902; Massalongo, *Rif. med.*, 1902; Giese-Schultze, *Z. f. N.*, 1902; Hödlmoser, *W. kl. R.*, 1903; Kollarits, *A. f. kl. M.*, Bd. lxxii.; Hun, *Albany Med. Annals*, xxv.; Diller, *Journ. Nerv. and Ment. Dis.*, 1903; Hey, *M. m. W.*, 1903; Mohr, *B. k. W.*, 1903; Sterling, *M. f. P.*, xvi.; Oppenheim, *D. m. W.*, 1904; Bilschowsky, *M. m. W.*, 1904; Pel, *B. k. W.*, 1904; Loeser, *Z. f. Aug.*, xii.; Léon, *Nouv. Icon.*, xvii.; T. Cohn, *N. C.*, 1904; Gowers, *Quart. Rev.*, 1904; Dupré-Pagniez, *Nouv. Icon.*, xviii.; Spiller, *Journ. Med. Sc.*, 1905; Bruns, "Ärtzl. Sachv.," 1905; Raymond-Alquier, *R. n.*, 1905; Steinert, *A. f. kl. M.*, Bd. lxxviii.; Steltzner, *A. f. P.*, Bd. xxxviii.; Curschmann-Hedinger, *A. f. kl. M.*, Bd. lxxxv.; Leclerc-Sarvonat, *R. n.*, 1905; F. Buzzard, *Br.*, 1905; Boldt, *M. f. P.*, xix.; Knoblauch, *A. f. P.*, Bd. xlii.; E. Levi, *Riv. di Patol.*, 1906; Albertoni, Bologna, 1906; Kauffmann, *M. f. P.*, xx.; Steinert, *A. f. kl. M.*, 1906; Muskens, *Tijd. vor Geneesk*, 1906; Sitsen, *B. k. W.*, 1906; Kétly, *Z. f. N.*, xxxi.; Raymond-Lejonne, *R. n.*, 1906; Marburg, *Z. f. Heilk.*, 1907; Kollarits, *Med. Klinik*, 1907; Borgherini, *N. C.*, 1907; Pel, *B. k. W.*, 1907; Delille-Vincent, *R. n.*, 1907; Tilney, "Neurographs," New York, 1907.

This peculiar disease has only been thoroughly investigated during the last 20 to 25 years. Our justification for appending it to bulbar paralysis rests upon the fact that bulbar symptoms occupy a prominent place in its symptomatology.

Erb (1878) was the first to note that there was a form of bulbar paralysis which differed from the well-known progressive form in its tendency to improvement, possibly to recovery. In the cases which he published he specially mentioned the occurrence of *ptosis and weakness of the muscles of the jaws and neck*. *Atrophy* and diminution of the electrical excitability were present. Although the importance which Erb ascribed to his cases was not derived from these factors, which have subsequently been seen to be the essential characteristics of the disease, there can be no doubt that his cases belonged to this class, and in view of this fact the affection has since been termed Erb's disease. His work did not, however, give the impulse to the investigation of this disease and to the establishment of our conception of it. A paper which I wrote in 1887 is of importance in this respect, as on the ground of a case which I had for a long time under clinical observation and had thoroughly investigated pathologically, I was able to demonstrate a new clinical condition of which the bulbar symptoms were a prominent feature. The

[1] Compare the corresponding data and figures in the section on "Infantile cerebral paralysis."
[2] *W. kl. W.*, 1899 and 1900.

most remarkable point was, however, that most careful microscopical examination of the nervous system gave a negative result (compare Figs. 382 and 384, which illustrate another case which I have since examined). I therefore considered myself justified in speaking of a *neurosis*, and indeed of a chronic progressive and fatal neurosis, which is mainly characterised by the symptoms of glosso-pharyngo-labial paralysis *without atrophy*. A case of bulbar paralysis without any pathological changes had been already described by Wilks, but with so little detail that is seemed impossible to classify it. My communication was soon followed by valuable analogous observations by Eisenlohr and others, and my assistant, Hoppe, then collected the corresponding cases, added a new case which had been under my observation and which I had diagnosed during life, and proved that we were dealing with a *special clinical condition*, a disease *sui generis*.

The essential characteristics which I had been already able to demonstrate in my case were the existence of *dysarthria, dysphagia, and weakness of mastication*, with corresponding *paresis of the muscles of the lips, tongue, palate, and jaws*. The *superior facial* was also involved, and closure of the eyelids was specially weakened. The paresis extended to the *muscles of the trunk and extremities*; there was *marked weakness* in the arms and legs, and *dyspnœa*. The *absence of atrophy and of the signs of electrical degeneration* seemed to me specially striking. These were not present even towards the end of life, although the illness had lasted 2¼ years. There were further to be noted the *marked fatigability* of the affected muscles, the tendency of the disease to *remissions*, and the absence of anæsthesia and of severe pain. Intercurrent attacks of great breathlessness, tachycardia, and rise of temperature occurred.

Later observations have added some other symptoms and have also made important contributions to our knowledge of the *course* of the disease. In this connection, Goldflam's cases are worthy of special note as they show that it may end in apparent *recovery*; that in any case it may disappear for months and years; that the symptoms may develop fully within a space of a few weeks or months, and that relapses may occur after remissions which have lasted for months and years. Goldflam has also shown that the cases described by Erb in 1878 belong to this group, and has proved that the *ocular muscles* are very often involved; that ptosis, in fact, is an almost constant symptom, and that this or a diplopia due to paralysis of the external muscles of the eyeball, or even ophthalmoplegia, may be the first sign of the disease, and may in a few cases (Karplus) be the most prominent symptom. Our own experience is that this ocular form of myasthenic paralysis is comparatively common. Finally, the fatigability of the muscles has been brought into greater prominence in the later cases. Thus in 1890 I observed in the Charité a young man who suffered from this disease, and who was unable to feed himself, because, although he could chew the first mouthful, he became so quickly tired that there was almost no muscular force in the closure of the jaws. The same fatigability became marked in the muscles of the extremities; he could take two steps in the usual way, then the gait became tottering and slow, and in a short time he entirely collapsed. *Electrical stimulation of the muscles* had the same effect; indeed, in an examination of the thoracic muscles, a condition of threatened *suffocation* ensued. The illness terminated fatally. An autopsy showed the nervous system to be intact and the muscles normal. Jolly made the microscopic exa-

mination and found negative results. He has also studied more closely the above-mentioned anomalies of the electrical excitability which I had previously described, and has given to them the name of "*myasthenic reaction*," whilst he has termed the whole morbid condition a "*myasthenia gravis pseudo-paralytica*." He has shown that during stimulation of the nerves or muscles by tetanising faradic currents repeated at intervals of seconds, the muscular contractions become weaker with each stimulation and in the end absolutely fail; but that after a short pause for recovery, the muscle regains its normal excitability. "If the current is continuously applied from a quarter to a whole minute, one observes a uniform diminution of the contraction, which sooner or later, according to the strength of the stimulation, completely disappears. Here also a pause of barely a minute is sufficient to make the stimulation again act in the same way." Murri found that the muscles exhausted by repeated faradisation still retained their normal voluntary excitability, but this is not the rule. On the other hand Curschmann and Hedinger showed that the muscles functionally exhausted by electrical stimulation gave way much more rapidly than those which had been resting. They state the surprising fact that a period of two seconds for recovery is sufficient to restore the excitability of the exhausted muscles. Muscles fatigued by the faradic, can still react to the galvanic current (Buzzard). The muscles and the nerves do not always show the phenomenon of electrical fatigability to an equal degree (Goldflam, Steinert). Further, exhaustion caused by electrical stimulation does not always lead to complete loss of the excitability, which again may only occur after prolonged stimulation. We have observed other modifications of the myasthenic reaction, which have been described by Sterling. I shall refer again to the question of the diagnostic and pathognomic value of the symptom.

The symptom of *fatigability* (myasthenia, apokamnosis) has since been brought into special prominence by Goldflam, Jolly, and Strümpell, who regard it as the cardinal symptom of the disease.

Grocco states that he found fatigability of the sphincter iridis. A kind of myasthenic action of the heart has also been described in a few cases, but X-ray examination by E. Levi showed nothing abnormal.

The *muscle tonus* may be normal; it is diminished in severe cases and in the stage of exhaustion. The hypotonia was very marked in a case described by Dupré-Pagniez, and this corresponds to an observation of Steinert's that the tendon reflexes may for a time disappear (his case, however, does not seem to me convincing). Raymond-Lejonne have found diminution of the blood pressure in one case, and this sign had already been noted by Bing in one of my patients, a case, however, which I felt bound to diagnose as polioencephalitis. Diller mentions vaso-motor disturbances. Cassirer and I have seen a case in which the attacks of paralysis in the eye muscles were always accompanied with chemosis, conjunctivitis, and exophthalmos.

Amongst the disturbances of sensibility, pain is not uncommon. It was exceedingly intense in a case treated by Buzzard. This writer also noted objective disturbances of sensibility, which I have never found in pure, typical cases. In these the sensory functions are almost always intact. Auditory disturbances have been found in a few cases (Oppenheim-Hoppe, Muskens).

To summarise we may say that the following are the characteristic symptoms of the disease: 1. In addition to the symptoms of bulbar paralysis there is weakness of the muscles of the trunk, particularly of the neck and extremities, and often of the external muscles of the eye (especially ptosis). The combination of lagophthalmus or of facial diplegia with the ptosis gives a characteristic look to the physiognomy

(Figs. 378-381). This form of bulbar paralysis is distinguished from the others by the marked and early accentuation of the weakness in the jaw muscles—the dysmasesia. 2. The muscles involved remain normal in size and electrical excitability. The reaction of degeneration is absent, but the myasthenic reaction is usually present. 3. The weakness is

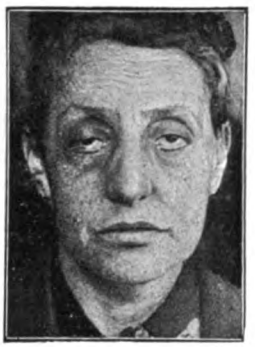


FIG. 378.—Facial expression in myasthenic paralysis. Combination of ptosis and facial diplegia. (Oppenheim.)

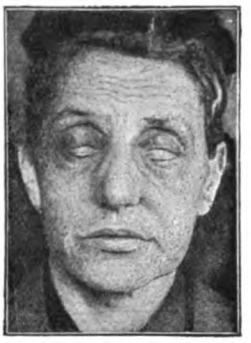


FIG. 379.—Same as Fig. 378, in attempt to close the eyes.

subject to marked changes and is associated with abnormal fatigability. This condition of exhaustion is sometimes evident in all the affected muscles, sometimes only in a few of them (see Figs. 380 and 381). 4. Sensory disturbances are as a rule absent, with the exception of slight

FIG. 380.—Case of myasthenic paralysis. Condition of levator palpebrarum and superior recti at commencement of upward movement of eyes. (Oppenheim.)

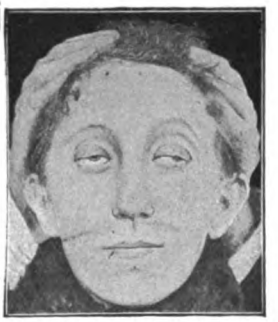


FIG. 381.—Condition of these muscles after the attempt to look up had lasted for a minute.

pain; and there are no disturbances of the functions of the bladder and rectum. 5. The mind is unaffected. 6. No pathological change is found in the nervous system, even when the disease has been in existence for years. Mayer found, by the use of Marchi's method, changes in the anterior roots, but I think it doubtful whether any importance should be ascribed to these changes or to those found by Marinesco-Widal, Murri, Sossedorf, and Fajersztayn, in the nerve-cells or in the intramedullary

root fibres. The case described by Dejerine and Thomas, which has not yet been fully published, is also uncertain. The changes described by Liefmann are also of doubtful value.

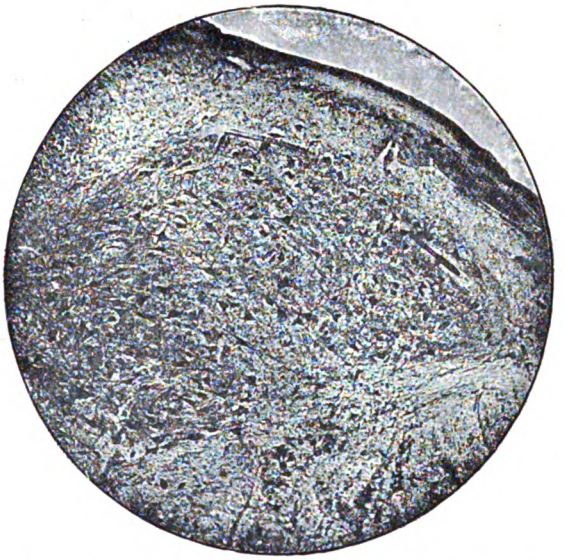


FIG. 382.—Hypoglossal nucleus in myasthenic paralysis (Carmin stain). The same section was given on p. 1008 as a type of the normal.

FIG. 383.—Compare with Fig. 382. Hypoglossal nucleus in progressive atrophic bulbar paralysis.

A few years ago Weigert stated the remarkable fact that in a case of myasthenic paralysis, he had found cell infiltration in the muscles, which he traced to a tumour in the thymus and was inclined to regard as

metastatic. This was soon followed by the communications of Goldflam-Flatau, Link, Hun, F. Buzzard, Boldt, and Steinert, who reported similar changes in the muscles, most of which, however, they interpreted in other ways. Buzzard's investigations were specially thorough and convincing; in every case he found the round cell infiltration, which he regarded as *lymphorrhages*, in the muscles, and possibly also in other organs (liver). He is not inclined to ascribe the functional disturbances directly to these foci, but regards them rather as merely the visible manifestation of a previous process of intoxication in the organism. He does not think that the relation of the infiltration to the thymus is yet explained. Knoblauch has found the same changes in portions of muscle taken from the living patient, and is of opinion (*D. m. W.*, 1908), that in myasthenia there is a preponderance of the " clear " muscle fibres over the red ones, whilst Marburg and Borgherini both found changes in

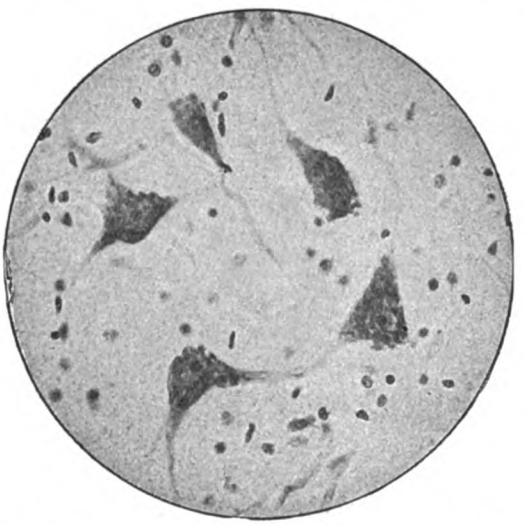


FIG. 384.—Cells from the hypoglossal nucleus of a woman who died of myasthenic paralysis Nissl stain.

the muscles which the former regards as a degenerative myositis, the latter as plasmoidal and hyaloid degeneration. In any case we must regard the *disease of the muscles* as the sole anatomo-pathological cause.

As regards the *course*, an acute or more often a subacute or chronic development of the symptoms is the rule; the further progress may be acute, chronic, or relapsing. In this way the disease may extend over twenty years. A few cases (Bernhardt, Pineles) show an intermittent, periodic course. The process may commence in the bulbar muscles, in the eye muscles, or in the muscles of the extremities.

As a rule young persons are affected, but there are exceptions.

The character and nature, the cause and origin of the disease is not even yet fully explained.

A *congenital predisposition* probably plays an essential part in its onset. Congenital anomalies of development have several times been found in the nervous system and other organs. In one case I found

micrognathia, in three polydactyly, and in another doubling of the great toe. Curschmann and Hedinger mention a combination with sexual infantilism. Of special interest in this respect is a condition found by Eisenlohr and confirmed by myself, viz., the presence of small fibres in the roots of the motor cranial nerves, and also the anomalies of formation in the Sylvian aqueduct which I found in one case (Fig. 385). The disease may apparently also be produced by infective diseases (influenza) and malignant tumours. Mediastinal tumours,[1] tumours or persistence of the thymus, were noted in several cases (Link, Hödlmoser, Dupré-Pagniez, Buzzard, Weigert, etc.), and in a case of Senator's which possibly belongs to this class, multiple myeloma. These probably act by introducing into the circulation products which have a toxic effect. We cannot

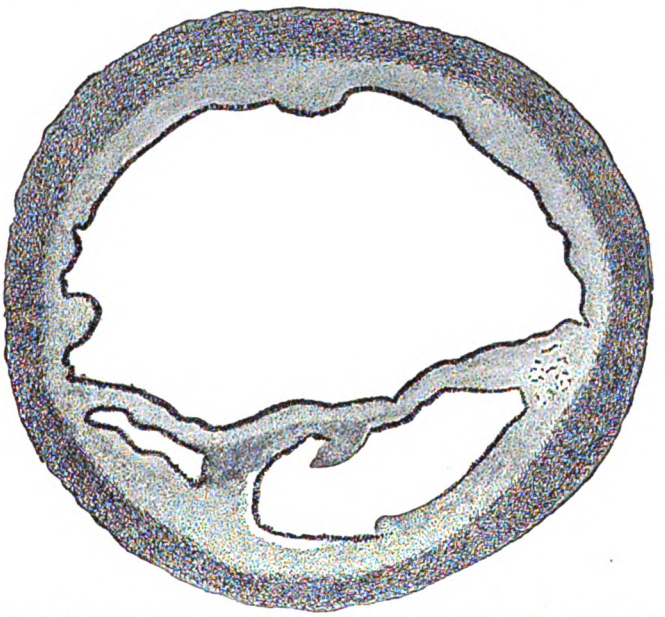


FIG. 385.—Anomaly of formation of the aqueduct of Sylvius, as found by Oppenheim in a case of myasthenic paralysis.

determine whether the co-operation of the two factors, the congenital predisposition, and the toxic impairment of the organism, is necessary to produce the disease. It is apparent from the changes described by Weigert, Goldflam, Link, Hun, Buzzard, and others, that a disease of the muscles may be thus produced. The fact that the muscle infiltrate arises from the remains of the thymus also points to the congenital origin. Chvostek (*W. kl. W.*, 1903) has suggested excess or loss of function of the epithelial bodies as the cause. Kauffmann, in his researches into its metabolism, found that the process was similar to that of acid poisoning, and thinks the autointoxication may arise from the liver, which he has found to be affected in several instances. Possibly several types may be distinguished according to the etiology, primary cause, and symptomatology.

[1] In a case of this kind under my observation the results of an X-ray examination were in favour of this view. Knoblauch has since corroborated this fact.

The *complication* with exophthalmic goitre and hysteria should be specially noted. Goldflam mentions the combination with facial hemiatrophy, Diller with angio-neurotic œdema, and Steinert with diabetes insipidus. In a very interesting case described by L. Mohr, the myasthenic paralysis was associated with Banti's disease. I have treated for this disease a young woman who had suffered from infantile spinal paralysis from her second year.

Some cases (Fuchs, Cohn, Oppenheim) point to the fact that myasthenia may be associated with dystrophy and possibly also with myotonia, but the nature of these mixed forms is still quite unknown.

As regards the *differential diagnosis*, the marked fluctuations and remissions may lead to a confusion with *hysteria*, but this is only possible on superficial observation and examination. The disease differs distinctly from the other forms of bulbar paralysis. The combination of bulbar symptoms with external ophthalmoplegia, usually incomplete, and with weakness of the muscles of the trunk and extremities, the development of these paralytic symptoms, which is not apoplectic nor acute but usually slow or relapsing, the marked accentuation of the weakness of the muscles of mastication and of the lagophthalmus, the frequent involvement of the neck muscles, the purely motor character of the paralytic symptoms, the preponderance of the myasthenia over the paralysis in some or in all of the muscles involved, the remittent course, the absence of true muscular atrophy and the corresponding changes of electrical excitability when the disease is of long standing, and the myasthenic reaction which is often present, all combine to produce a characteristic picture, absolutely different from the type of the other bulbar diseases. Forms of reaction related to the myasthenic have certainly been described in other diseases (Benedict, Brenner, Oppenheim, Flora, Kollarits, Curschmann, Hedinger, Steinert), but it is not proved to occur in its typical development in other affection. Steinert's statement that the myasthenic reaction always accompanies hemiplegia urgently requires testing. I have myself been in doubt as to the interpretation of this reaction, as I have examined from this point of view *fifteen healthy soldiers and in not a few of them found that the contractions, especially in the deltoid, became weaker after five to six stimulations*, which would certainly have previously suggested to me the myasthenic reaction. Certainly the reaction was never entirely lost, but that is very often the case in myasthenic paralysis also. We therefore require more observations taken with care, and great caution should be exercised in their interpretation. This is the more to be regretted, as in the initial stages and in the abortive forms of the disease it would only be possible to give a definite diagnosis if the theory of the myasthenic reaction in Jolly's sense could be maintained.

As regards the basis of the myasthenic reaction, see Salomon (" Polioclinico," 1907).

It is most difficult to distinguish myasthenic paralysis from *polioencephalomyelitis*. But in the chronic form mainly concerned there is always marked atrophy with great diminution of the electrical excitability, or less often with reaction of degeneration. Nor are the marked remissions and signs of fatigue present in this affection.

Symptoms of a bulbar paralysis of a non-myasthenic character, may develop in malignant tumours, without corresponding changes being found in the medulla oblongata (Nonne), or as the result of processes of degeneration (Hensen, Osann). This is also the case (as already described on

p. 1019) with regard to the symptoms in typhoid, in meat, sausage, and fish poisoning (p. 832), and in poisoning with barium salts (Fajersztayn). There is no doubt that bulbar paralyses of toxic origin which are not identical with myasthenic paralysis exist without any pathological changes.

In the cases observed by Jolly and myself there was much to remind one of *muscular dystrophy*, which had also been diagnosed by other physicians, but the dystrophy and the corresponding condition of the electrical excitability were not present. Gowers also notes the similarity. The disease has a superficial likeness to Gerlier's disease (vertige paralysant; see chapter on vertigo). Finally we should remember that there is a form of Landry's paralysis (*q. v.*) which commences in the muscles innervated by the medulla oblongata, and which may terminate fatally before the extremities have become paralysed.

The *prognosis* is grave. There is always danger to life, and out of thirty-eight cases twenty-six ended fatally. Improvement and even recovery is, however, not impossible, and even the most severe symptoms of paralysis may disappear. But after a stage of complete health they may suddenly reappear and lead to a fatal termination. Goldflam specially notes the possibility of recovery. I have seen a remission of nine years, and Raymond-Sicard regarded one case as having been cured four years previously.

Treatment of these cases requires great care. In the first place I would advise avoidance of *stimulating electrical treatment*, and of the ordinary faradic or galvanic stimulation of the muscles, such as we are accustomed to use in other kinds of bulbar paralysis. On the grounds already mentioned, I regard these measures as dangerous to life in this disease. On the other hand central galvanisation is permissible and appropriate.

The most important requisite is to spare the affected muscles from all fatigue. In severe cases and attacks, the patient should keep to bed, and should avoid any movement of the muscles. He should speak very little. In one advanced case to which I was called, the adoption of these measures led to marked improvement, but I made a mistake in one point. I was desirous of saving the patient from swallowing, and recommended that he should be fed by the tube, without reflecting that the gulping movement elicited by the introduction of the tube might involve much more exertion than the movements of mastication and swallowing involved in natural feeding. The patient died in an attack of suffocation during artificial feeding. It is, therefore, obvious that a feeding-tube should only be used in cases of this kind when it goes smoothly down and provokes no spasmodic movements. Bruns recommends that narcotics should not be used. Careful feeding is of the utmost importance, and food should be given in such a way as to avoid as far as possible all fatigue of the muscles of mastication and deglutition (semi-solid foods of a very nutritious nature, intervals for rest between the meals, with complete physical rest before and during them).

Tonics are also suitable. Since Weichardt (*M. m. W.*, 1904) has made his interesting investigations upon the toxins and anti-toxins of fatigue, I have also made experiments in cases of this kind with the latter, which Weichardt has been good enough to send me, but the results have not been satisfactory. Kauffmann recommends as a palliative measure, subcutaneous injection of a double salt of spermin and sodium chloride, giving one c.cm. of a 2 per cent. solution daily or every second day. Delille-

Vincent reports a remarkable improvement in one case from the use of pituitary and ovarian preparations.

I think it not impossible that in the future the disease may in some cases be cured by the removal of a tumour which is the source of the toxic products. Tumours have already been successfully removed from the anterior mediastinum (Türk).

It should also be remembered that treatment of mediastinal tumours with X-rays has been recommended by Kienböck (*W. m. Pr.*, 1903), Clopatt (*D. m. W.*, 1905), Elischer-Engel (*D. m. W.*, 1907), but this measure has always failed me in myasthenic paralysis

Ophthalmoplegia

Literature: this is collected in Wilbrand-Saenger, and in an exceedingly careful study by Uhthoff in Graefe-Saemisch, "Handbuch der Augenheilkunde." 2nd edition. The article by Uhthoff should specially be referred to. Of the more recent contributions see Axenfeld, *C. f. N.*, 1905; Altland, *A. f. Aug.*, Bd. xlix.; Chaillons-Pagniez, *Nouv. Icon.*, 1905; Bramwell-Sinclair, *Scot. Med. and Surg. Journ.*, 1906.

Paralysis of the ocular muscles occurs under very various conditions and is a symptom of numerous diseases (see p. 465). There are, however, some diseases in which bilateral paralysis of the eye muscles is so very prominent, that it forms the most important symptom or may indeed be the only one. Although it is hardly possible to differentiate between the cases in which the ophthalmoplegia indicates an independent disease and those in which it is only one among other symptoms of a disease, yet we are apparently justified in recognising it is a nosological entity.

Ophthalmoplegia may develop in an *acute*, *subacute*, and *chronic* way. (The chronic form may have a remittent or an intermittent character.)

Acute ophthalmoplegia is an affection which usually arises from some infection or intoxication. Our knowledge of its pathological cause is defective. A hæmorrhagic encephalitis—the poliencephalitis acuta hæmorrhagica superior described by Wernicke—is undoubtedly the cause in a great many cases (see p. 834). But there are also toxic forms of acute ophthalmoplegia without any evident lesion, and it may be difficult in given cases to decide whether we are dealing with a simple intoxication paralysis or an encephalitis, the more so as in the latter also, at least in the form localised in the central grey matter, the disproportion between the symptoms and the comparatively slight changes may be remarkable (Boedeker, Hoffmann).

Chronic alcoholism is the principal cause of these conditions, but other poisons, such as lead, carbonic oxide, sulphuric acid, bisulphide of carbon (?), ergotin, and phosphorus (Medea) may produce paralyses of the same nature.

The literature contains a great number of cases of *ophthalmoplegia* of acute onset with or without *bulbar symptoms*, which in the absence of pathological examination cannot be definitely interpreted. Most of these cases had a *toxic* or *infective* origin. The majority of the ophthalmoplegias caused by meat, sausage, and fish poisoning, and a great many of those due to acute infective diseases, such as influenza, must be included in this group with an indefinite pathological basis.

The ophthalmoplegia of *botulism*, according to the classification of Wilbrand-Saenger, Lewin-Guillery, and Uhthoff, has a decided tendency

to affect the internal muscles of the eye, a bilateral internal ophthalmoplegia being the rule. The levator palpebræ superioris is next most commonly affected.

The nuclear ophthalmoplegia which is related to *syphilis* may develop acutely, but it usually assumes a chronic form and will therefore be discussed later.

The affection may undoubtedly be produced by a simple *traumatic* hæmorrhage in the central grey matter. Traumatic late apoplexy (see p. 795) may specially have this effect. Thus I have seen an ophthalmoplegia and paralysis of the muscles of mastication appear in a boy shortly after a fall on the occiput, a condition which could most easily be explained on the assumption of a hæmorrhage in the central grey matter in Bollinger's sense. There was, however, a hereditary predisposition, as the mother, grandmother, and three aunts of the boy suffered from congenital eye troubles (myopia, strabismus, capsular cataract, and ocular paralysis). Luce has subsequently given the same explanation to a case of this kind and has sought to apply it to all cases of "infantile nuclear atrophy."

Cases of traumatic ophthalmoplegia interna (P. Simon, Tumpowski, Schultze), and of reflex immobility of pupils of traumatic origin (Axenfeld, Dreyfus), have also been described, but they are few and uncertain.

There is no doubt that many affections of the pons or corpora quadrigemina, *e.g.* new growths, and in particular solitary tubercle, may produce opthalmoplegia.

The chief points as regards the symptomatology of so-called nuclear ophthalmoplegia have been already discussed (p. 469), so no further description need be given here. We have also already referred to the objection recently raised by Bach, that the signs hitherto regarded as criteria of nuclear affections can no longer be taken as absolute, and that even pathological examination affords no certain evidence, in so far that the lesions of the roots and nerves may produce secondary changes in the nuclei.

The ocular paralysis caused by morbid processes at the base of the brain, *e.g.* tubercular meningitis (Oddo-Olmer) and syphilis (Uhthoff, Oppenheim), may correspond to total ophthalmoplegia.

It should further be remembered that acute ophthalmoplegia may be the result of a peripheral neuritis of the ocular nerves (Dammron-Meyer, Dejerine, etc.), and that there are absolutely no trustworthy means of differentiating the neuritic from the nuclear form.

Unilateral nuclear ophthalmoplegia is apparently rare. If it involves the entire oculo-motor nucleus, we should expect one or more of the ocular muscles on the same side to be intact, and the corresponding ones on the opposite side to be paralysed. So far as I can see, only one case, that of Wishart (*Journ. Nerv. and Ment. Dis.*, 1897), corresponds to this postulate, as in it the inferior oblique was only paralysed on the opposite side.

For the differential diagnosis we would refer to the description of "bulbar paralysis without pathological change" in the foregoing chapter. An ophthalmoplegia unaccompanied by pathological changes has also been found in exophthalmic goitre (Bristowe, Warner, Ballet, Rothmann, Liebrecht). It has already been said with sufficient emphasis that nuclear ophthalmoplegia is often associated with bulbar symptoms.

The prognosis of acute ophthalmoplegia is doubtful. In the form due to poliencephalitis of *alcoholic* origin it seems to be most unfavourable.

but even there recovery is possible. If it is caused by influenza or follows other infective diseases and intoxications, recovery is frequent; indeed, it follows in the majority of such cases.

Chronic ophthalmoplegia, which has also usually a *progressive* character, is seldom observed in its pure form. We owe the first description of it to von Graefe. The disease develops as a rule in the following way: the patient becomes affected with diplopia or ptosis, very rarely with associated ocular paralysis. Very gradually, in the course of many months or years, the paralysis spreads to *all the ocular muscles of both sides*, in such a way that the internal muscles of the eye, the sphincter iridis and ciliary muscle, as well as the levator palpebræ superioris, are often or usually spared (Uhthoff). This form of *ophthalmoplegia exterior bilateralis* bears clearly the stamp of its nuclear origin. It would be difficult to ascribe a paralysis that develops in this manner to a peripheral process, whilst the fact that the oculomotor nucleus is composed of cell groups, which apparently represent the centres of the various ocular muscles (see p. 652), makes it easy for us to understand the nuclear origin of an ophthalmoplegia exterior. It is not uncommon, however, for the internal ocular muscles to be involved either from the first or later, and Monakow points out that in the cases in which a nuclear disease was pathologically evident, the internal muscles were usually involved.

In the advanced stages the eyeballs become quite immobile, with their axes either parallel or slightly divergent; they respond to no voluntary impulse. The levator palpebræ is the only muscle that is usually incompletely paralysed. In a few cases the orbicularis palpebrarum was also involved. These symptoms may in themselves constitute the whole of the disease, and the process, after having in the course of months, years, or decades led to total paralysis of the eye muscles (in Altland's case the development took thirty, in Beaumont's thirty to forty years), may come to a termination. Strümpell reports a case of this kind in which the paralysis has been stationary for fifteen years.

It is surprising that in none of the cases hitherto examined post-mortem had the affection been limited to the nuclei of the ocular nerves, as Siemerling states; and clinical experience also shows that in the majority of these the chronic ophthalmoplegia is merely the *precursor* or the *local symptom* of a *complicated* disease of the central nervous system, such as *tabes dorsalis, general paralysis, combined column diseases of the spinal cord, disseminated sclerosis*, an atypical form of *progressive bulbar paralysis*, and even *progressive muscular atrophy* or chronic anterior poliomyelitis. Mixed forms of ophthalmo-bulbar paralysis with the symptoms of degeneration of the brain tracts have been described. Wilbrand and Saenger have collected and reviewed all the published cases.

They are specially apt to be associated with symptoms of tabes dorsalis, and indeed five to seven years and more may elapse before these appear. The combination with general paralysis has also been often observed, and it is worthy of note that the disease tends very much to be associated with mental symptoms (Westphal, Siemerling). The cases in which the affection passes to a certain extent from above downwards, *i.e.* from the nuclei of the ocular nerves to those of the bulbar nerves, are of special interest. In such cases the facial of both sides is usually first affected, then the muscles of the tongue and palate, until finally marked symptoms of bulbar paralysis appear. The disease may be unaccom-

panied by pathological changes (see preceding chapter), or it may take the form of so-called poliencephalitis superior et inferior. Marked and often exclusive involvement of the upper facial has been specially noted in cases of the first class, but is perhaps not confined to them. If an atrophic paralysis of the extremities supervenes, we have the picture of *chronic poliencephalitis*, as it has been described by Rosenthal, Seeligmüller, Eichhorst, Bristowe, Sachs, Guinon-Parmentier, Schaffer, Bernhardt, Roth, etc. But the process may commence at any point, and ascend or descend.

Finally, it should be noted that ophthalmoplegia may be a congenital disease or may be early acquired on a hereditary familial basis. Cases of this kind have been described by Gräfe, Baumgarten, Steinheim, Mauthner, Hirschberg, Uhthoff, Moebius, Schapringer, Kunn, Heuck, Heubner, Cabannes-Barneff, Péchin, Chaillons-Pagniez, etc. In these there was sometimes merely paralysis of single ocular muscles, especially the levator palpebræ superioris and the abducens, sometimes a more or less complete ophthalmoplegia which, however, spared the internal muscles, the sphincter iridis in particular, or on the other hand was sometimes associated with paralysis of other cranial nerves, *e.g.* the facial, and other malformations.

Observations by Reichardt, Finkelnburg, and myself leave no doubt that in very rare cases immobility of the pupils is a congenital disease. Levinsohn (*B. k. W.*, 1907) has also diagnosed a congenital origin in a case of bilateral ophthalmoplegia interna.

Few examinations have hitherto been made into the pathological cause of this congenital affection. Moebius assumes that there is a defective development or an actual absence of the nuclear region (" infantile nuclear atrophy "). Heuck in one case found the muscles atrophied or degenerated, the nerves being macroscopically intact; he suggests a primary muscular atrophy. The muscles may, however, be histologically intact in congenital paralysis of the ocular muscles (Heuck, Axenfeld). Siemerling, in a case of congenital ptosis, found a nuclear defect, and Heubner was able to show by pathological examination of his case that it was due to an aplasia of the corresponding nuclear region. This ophthalmoplegia arising from a congenital predisposition may appear as a family disease after birth and even in adult age (Dutil, Beaumont, Homén, [the latter observed it in twins [1]] Delord). It is not therefore always a complete aplasia, but possibly a hypoplasia, a defective formation (Marina), a " congenital inferiority " (Oppenheim), an " abiotrophia " (Gowers) of the corresponding nuclear apparatus which causes its premature death and decay.

I have noticed that an injury to the head may under such circumstances give rise to the development of the disease. We can only refer briefly to the exceedingly interesting cases of Axenfeld (*C. f. N.*, 1905), in which " cyclical congenital disease of the oculo-motor nerve " was associated with rhythmic spasms in the muscles supplied by them (see p. 468).

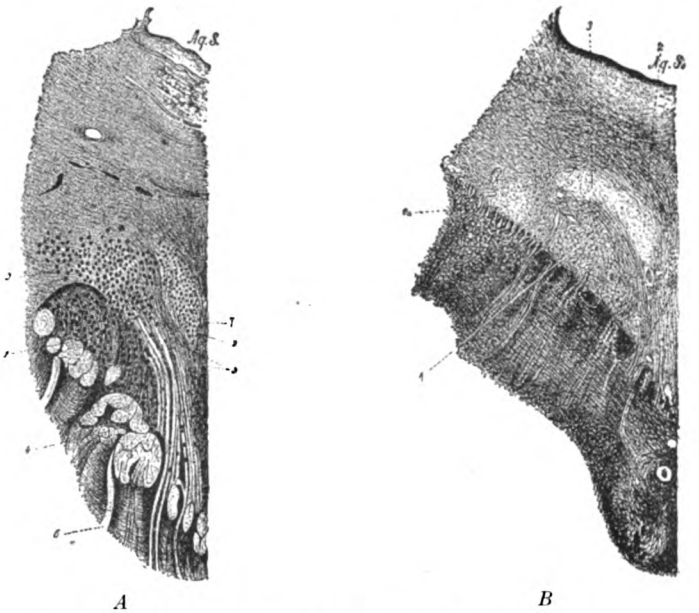
I have shown that degeneration of the nuclei of the ocular nerves may develop from *syphilis*, but our knowledge of the so-called nuclear ophthalmoplegia of this origin is still defective The disease was attributed to hereditary syphilis in one case by Wilbrand-Saenger, and in

[1] His case is also interesting from the fact that he performed transplantation of the frontalis to the levator palpebræ superioris to compensate for the ptosis.

another by Cabannes. We should also refer to the possibility of a traumatic origin.

The *prognosis* of chronic ophthalmoplegia is on the whole very unfavourable. In a very small proportion of cases the disease may improve spontaneously, or, if it is due to syphilis, under treatment. More often it becomes stationary, but as a rule the symptoms of a disease of the brain or spinal cord supervene, and the patient is threatened with this danger even after the lapse of years.

Chronic ophthalmoplegia is frequently associated with atrophy of the optic nerve, either as a symptom of a primary tabes dorsalis or general



A *B*

FIG. 386.—*A* and *B*. (After Siemerling.)

A. Normal oculo-motor nucleus.
1. Ventral oculo-motor nucleus.
2. Medial Group of III.
3. Lateral Group of III.
4. Posterior longitudinal bundle.
5 and 6. Root fibres.
7. Raphe.

B. Atrophy of the oculo-motor nucleus.
1. Ventral oculo-motor nucleus.
1a. Dorsal oculomotor nucleus.
2. and 3. Westphal-Edinger groups.

paralysis, or as the only complication. Persistent headache and pain in the eye have been noted in some cases, and have been ascribed to involvement of the cerebral trigeminal root (?).

The *pathological cause* of this disease (chronic ophthalmoplegia) is always, or almost always (especially according to the investigations of Westphal-Siemerling [1]), a *chronic inflammatory* or *degenerative process* on the floor of the third ventricle and of the Sylvian aqueduct, which terminates in destruction of the nerve-cells in the nuclei (compare Fig. 386, *B*). To all appearance the disease is a primary one of the nerve-cells,

[1] Previous to them Hutchinson and Gowers, and since then Boettiger, Oppenheim, Boedeker, Pacetti, Marina-Zeri, Cassirer-Schiff, and others have found similar conditions, and Siemerling and Boedeker have collected a large number of new cases.

which subsequently involves the whole nucleo-peripheral neurone. The roots and the peripheral nerves and muscles are degenerated according to the affection of their trophic centres.

These changes may be associated with those of tabes dorsalis, paralytic dementia, progressive bulbar paralysis, etc., which may, however, be but slightly developed.

Teratment.—In cases of acute onset antiphlogistic or diaphoretic measures, viz., ice-bags, blood-letting in the temporal region, aperients, hot packs, are suitable. I have seen remarkable results from the last-mentioned treatment. If syphilis is the cause, antisyphilitic treatment should be adopted. This is also the method to be followed in the chronic form, if syphilis is in question. Otherwise treatment should be directed to the primary disease. Electrical treatment has practically no effect, although galvanic treatment has been greatly recommended by some physicians. In a few cases under my care improvement followed a course of bathing at Oeynhausen.

As regards surgical treatment of ptosis, the text-books on ophthalmology should be consulted.

DISEASES OF THE CEREBELLUM

The morphological dissection of the cerebellum into a number of segments, labelled with special names, has hitherto been of no real value to pathology, although some data as to the relation of the various parts to the muscles of certain segments of the body have lately been given, especially by Bolk and Rynberk. Our knowledge of the functions of the organ as a whole is still defective, although it has been greatly advanced of late years by the investigations of Ferrier, Luciani,[1] Lusanna, Jackson, Horsley,[2] Russell, Bruce,[3] Thomas,[4] Bruns,[5] Probst,[6] Kohnstamm,[7] Boek,[8] Munk,[9] Bing,[10] and others. As regards its anatomical connections the description given on pp. 642 *et seq.* should be consulted.

The results of experimental investigation and clinical pathology harmonise in many respects ; the latter are for us decisive.

The cerebellum has no relation to the sensory functions and to conscious sensation. The statements of Probst, Kohnstamm, Lewandowsky (*Du Bois Arch.*, 1903), and Munk are only in apparent conflict to this statement. The cerebellum has a predominant influence on muscular movements. The disturbances which follow lesion of the cerebellum consist in inco-ordination of the movements. This becomes specially apparent in standing and walking. In standing the patient begins to sway ; he stands with his legs wide apart, and his uncertainty can be recognised from the rocking movements which are produced by contraction of the extensors of the foot and toes. A high degree of this inco-ordination makes standing quite impossible, and even sitting may become difficult or impossible without support on account of the swaying of the trunk. The unsteadiness is not usually increased when the eyes are closed, though it may be so. The *gait* is like that of a drunken man ; walking may even become quite impossible in the severest cases. The legs are affected to a greater extent than the arms. Indeed, according to Munk and

[1] " Il Cerveletto," Frienze, 1895.
[2] Clarke-Horsley, *Br.*, 1905, and Horsley, *Br.*, 1907.
[3] *Brit. Med. Journ.*, 1899.
[4] " Le Cervelet," Paris, 1897.
[5] *W. kl. R.*, 1896, and *B. k. W.*, 1900.
[6] *A. f. P.*, xxxv.
[7] *Pflüg. A.*, Bd. lxxxix.
[8] " De physiol. beteek. von het cerebellum," Haarlem, 1903. See also Rynberk, *Arch. di Fisiol.*, 1904.
[9] *Sitzungsber. d. K. P. Ak. d. Wiss.*, 1906.
[10] " Die Bedeutung der spino-zerebell. Syst.," Wiesbaden, 1907, with bibliography.

Bing, cerebellar inco-ordination is almost entirely limited to the muscles of the pelvic girdle and the lower extremities. In simple movements of the limbs, *e.g.* in raising the leg in the horizontal position, the ataxia is usually not present, but diseases of certain portions of the cerebellum may give rise to a true motor ataxia (especially in the arms).

Babinski (*R. n.*, 1899) points out that the synergy which normally exists between the action of the muscles of the trunk and extremities (leg) is relaxed by affections of the cerebellum, so that, *e.g.* in walking, the legs push forwards but the trunk remains behind, and *vice versâ*, in bending the head and trunk backwards the legs do not flex in the normal way; in rising from the horizontal position the legs are raised instead of the trunk, etc. ("cerebellar asynergy"). This disturbance may be limited to one side of the body ("hemiasynergy"), and is then probably due to a disease of the cerebellar hemisphere, the restiform body or Deiters' nucleus of the same side, and of the corresponding tectospinal tract. Babinski himself, however, regards this point as still hypothetical. Compare the papers of Laignel-Lavastine (*Nouv. Icon.*, xix.) and Rossi (*Nouv. Icon.*, xx.).

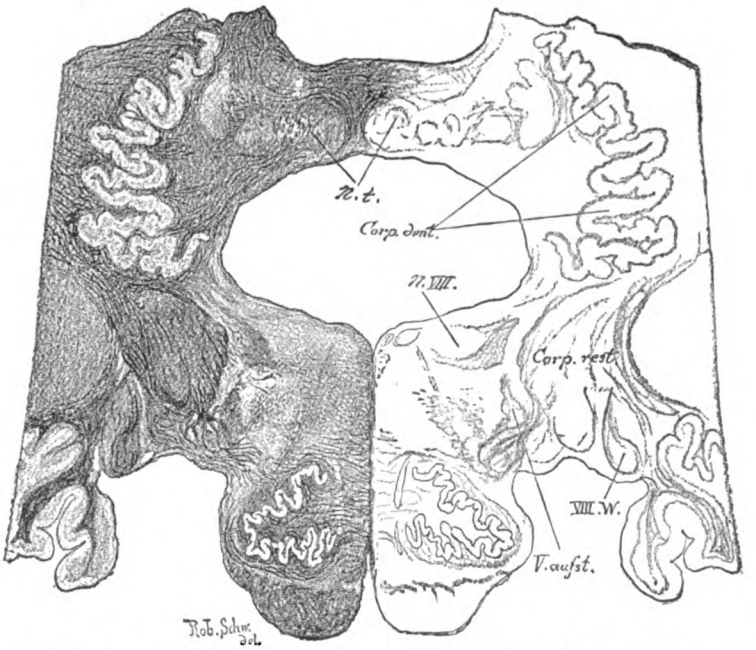


FIG. 387.—Section through medulla oblongata and adjacent part of the cerebellum. (Weigert preparation.)

Babinski (*R. n.*, 1902) has drawn attention to two additional cerebellar symptoms, the diagnostic value of which does not appear to me to be yet established. He states that in diseases of the cerebellum the power to maintain the limbs in a certain state of equilibrium, *e.g.* when in the horizontal position to keep the hip-joint flexed and the leg abducted—it not only conserved, but may be exaggerated, so that after short oscillations a fixation appears which is more firm and steady than in normal conditions. He terms this condition cataleptic. This factor radically distinguishes cerebellar from tabetic ataxia. By "diadococinesis" Babinski (*R. n.*, 1902) understands the power to carry out a number of antagonistic movements, *e.g.* pronation and supination, in rapid succession. In diseases of the cerebellum this may only be affected in such a way that although the strength is fully conserved, the rapidity of the successive movements is diminished (adiadococinesis). This symptom appears from our experience also to be a valuable one.

Diseases which are limited to the cerebellum may be latent throughout their course. Irritative processes which have their seat in the cerebellar hemisphere or in the middle cerebellar peduncle, may be manifested by *forced attitudes* and *forced movements*. Rotation or rolling round the axis of

the body (which, however, is not very important in human pathology), and the tendency to fall to one side, are specially characteristic. The latter is usually a symptom of paralysis. In diseases of the restiform body the patient usually falls towards the side of the lesion. Abnormal positions of the eyes have been noted in these conditions.

Paralysis of the body in the form of *homolateral* or *crossed hemiplegia* is not uncommon in cerebellar diseases. This is probably not a direct result of the cerebellar lesion, as it occurs almost exclusively in diseases which cause compression of the brain. There is no doubt that *compression of the pyramidal tract* in its course through the pons and oblongata is the cause of this

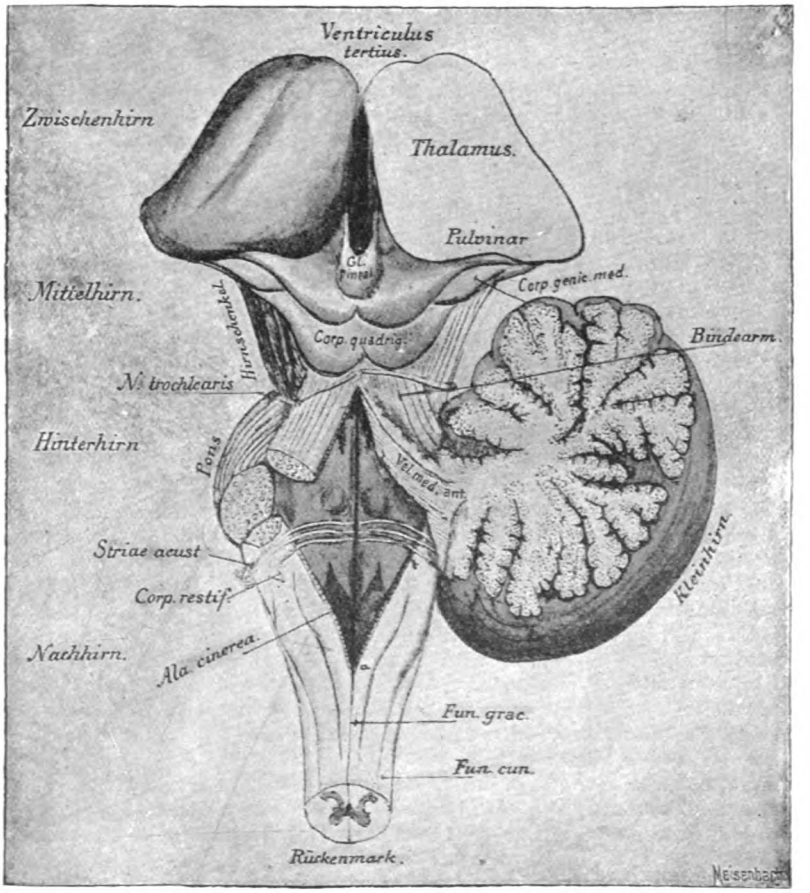


FIG. 388.—Mid and Hind Brain, exposed by removal of their roof. Cerebellar peduncle visible. (After Edinger.)

paralysis, and that it depends upon the site of the compression, above or below the pyramidal decussation, whether the hemiplegia develops on the side of the cerebellar disease or the opposite one. It has then the characteristics of spastic paralysis, but these may apparently also be compensated by simultaneous lesion of other tracts. Ferrier, Luciani, Horsley-Loewenthal, Bianchi, Probst, Personali, and a few clinicians such as Hitzig and specially Mann (*M. f. P.*, xii., see also xv.), attribute to the cerebellum an influence upon the strength of the muscular efforts of the homolateral side of the body; and the anatomically defined tracts which pass from the cerebellum, either directly or by way of the vestibular nucleus, into the homolateral side of the spinal cord apparently justify this view. I must conclude, however, from my own experience and the published cases of other writers (from Mann's description also), that we are here dealing not with a paresis, but with a *hemiataxia*, which is practically identical with Babinski's hemi-

asynergy. Bruns (*N. C.*, 1904) agrees with me. He almost always found the homolateral ataxia in the arm. Probst does not speak of paralysis, but of flaccidity and ataxia, and the atonic character of the paresis is noted by Mann himself. The fact that this writer ascribes the symptom to the absence of centripetal stimulations shows that the idea of paresis is not in his mind. Saenger (*N. C.*, 1907) appears to agree with Mann.

The *motor symptoms of irritation*, which occur chiefly in cerebellar diseases which cause compression (general, unilateral convulsions, tremor, clonic spasms), are probably not the result of the disease of the cerebellum but of irritation of the motor structures or centripetal tracts in its neighbourhood. But the chorea or hemichorea and the allied phenomena have been traced by some writers directly to lesion of the cerebellum, mainly of the vermiform process or the superior peduncle (and its processes) (Bonhoeffer, etc.; compare p. 693).

The peculiar attitudes and the conditions of tonic spasm (opisthotonus, etc.) described by Jackson probably occur mainly in acute destructive or chronic processes which cause compression, but this question requires further investigation. Buzzard's case (*Br.*, 1906) from which he infers a relation of the dentate nucleus to this special attitude, is not sufficiently convincing. The homolateral spasms observed in rare cases of cerebellar affection appear to occur only in tumours. As regards their causation, an article by Weber may be consulted (*M. f. P.*, xix.). I have attributed the rhythmic spasms of the muscles of deglutition and of the larynx, observed by myself and others (see p. 680 and 906) in diseases of the posterior cranial fossa, to "distant action," whilst Klien (*N. C.*, 1907) is inclined to relate them to the cerebellum itself. The attempt of Kleist (*Journ. f. P.*, x.) to connect the spastic movements sometimes noted in cerebellar affections, and which remind one of the persistent contraction in myotonia (?) with a lesion of the cerebellum, and specially of the frontal pontine-cerebellar tract, and to relate the "oscillating swinging tremor" also to these parts, seems to me to be unsuccessful.

Vomiting is not a direct cerebellar symptom, and is probably related rather to the medulla oblongata. This is the case also as regards the symptoms of irritation and paralysis of the motor cranial nerves. This is doubtful, however, as regards the *nystagmus*, which is not uncommon in cerebellar diseases. Perhaps it is also a pressure symptom, although the relations of the cerebellum to Deiters' or the vestibular nucleus,[1] and through these to the nuclei of the ocular nerves, seems to point to the possibility of the nystagmus being a direct result of the cerebellar disease. Experiments by stimulation have also been interpreted in this way (Wertiloff). Some writers, *e.g.* Bruce, Wallenberg, Adler, Probst, Babinski-Nageotte, include nystagmus and paralysis of conjugate deviation (towards the side of the focus) among the direct symptoms of lesions of the cerebellum. Horsley has also assumed relations of this kind. Nevertheless I do not regard true paralysis of conjugate deviation as a cerebellar symptom.

The influence of the cerebellum upon muscle tonus is still doubtful, but diseases or destruction of it seem to have a hypotonic influence upon the muscles of the same side of the body, which may declare itself in weakness of the tendon reflexes. I am doubtful whether this may, as some writers think, amount to actual absence of the reflexes. In any case Babinski's and Oppenheim's signs are not observed in diseases which do not extend their influence beyond the cerebellum. The origin of the intention tremor which has been repeatedly noted, especially in affections of the cerebellum which cause compression, is also unexplained. I think it probable that there are parts of the cerebellum, or of the tracts which arise in it, lesion of which may give rise to a form of tremor akin to intention tremor.

Mental disorders have been several times described in stationary diseases of the cerebellum, but some complicating disease of the cerebrum, especially hydrocephalus, etc., may perhaps have been always present.

The *speech affection* which occurs in cerebellar diseases, described sometimes as dysarthria or scanning, sometimes as ataxic speech, may be dependent on the effect of the process upon the medulla oblongata or on a simultaneous disease of the latter. It is, however, not improbable that the co-ordinating effect of the cerebellum extends to the muscles of articulation, so that a lesion of this organ may result in inco-ordination of the speech movements.

The direct cerebellar symptoms therefore are: *inco-ordination, cerebellar ataxia, homolateral motor ataxia, vertigo,* and probably a form of *speech disturbance* and *nystagmus*. They also

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[1] This name is applied to the whole group of nuclei, as we cannot here discuss the precise anatomical and physiological differentiation of the nuclei of Deiters and Bechterew, the angularis, triangularis, etc.

include *Babinski's asynergy*, if a separate place be ascribed to it, a very inconstant form of tremor or shaking, and *adiadococinesis*.

Jackson and Russell (Alder also) would attribute to the cerebellum, as Schiff and others had already done, a special influence on the muscles of the trunk. This is right only in so far that it plays a prominent part in the maintenance of equilibrium, and that the impulses from the lower extremities and the trunk are conveyed to the cerebellum mainly by the spino-cerebellar tracts. I have also seen in severe cases of cerebellar disease a disturbance of the innervation of the trunk muscles, which caused a sinking down and absence of erectness of the body.

How does the cerebellum fulfil its co-ordinating functions? Bruns, following Bechterew and more especially Bruce, has attempted to explain this process. The question has of late years been studied by Probst, Kohnstamm, Mann, Lewandowsky, and experimentally by Orestano (*Riv. di Patol.*, 1901), Pagano (*Riv. di Patol.*, 1902 and 1904), Prus, Clarke-Horsley, Marassini (*Arch. di Fisiol.*, 1905), Lurié (*N. C.*, 1907), and others, and their teaching agrees on the main points. The cerebellum receives from the skin, muscles, joints, semicircular canals, and other peripheral parts, impulses which regulate its activity. The centripetal tracts concerned are the direct cerebellar tract, Gowers' tract, the tracts connecting the nuclei of the posterior column with the cerebellum, the vestibular nucleus, all of which, according to Bruce and others, terminate in the vermiform process. They possibly include also the central bundle of the tegmentum and the cerebello-olivary tract,[1] and the fibre bundles which practically run in the restiform body. By means of these, stimuli flow to the cerebellum and inform it as to the position and attitude of the limbs, trunk, head, and eyes, and to some extent as to the condition of tension of the muscles. The cerebellum must further act upon the muscles of the trunk and extremities, either directly or by means of the motor centres, or in both these ways. A bundle of fibres passing in the middle cerebellar peduncle to the pons and thence into the ground bundle of the antero-lateral tracts was supposed to be a *direct* motor tract, but this connection has been discovered by recent investigation to be effected by tracts which reach the spinal cord by means of Deiters' or the vestibular nucleus (*tractus vestibulo-spinalis*, Bruce; see Fig. 389). According to Clarke and Horsley the tracts go from the cerebellar cortex to the roof nucleus and thence to Deiters' nucleus, etc. Other tracts are contained in the superior cerebellar peduncle, which by means of the optic thalamus, etc., connects the cerebellum with the cerebrum. Although these are essentially centripetal bundles, it is not impossible that stimuli influencing the motor centres may reach the cerebrum in this way. Finally, we must bear in mind that the cerebrum may interfere with the

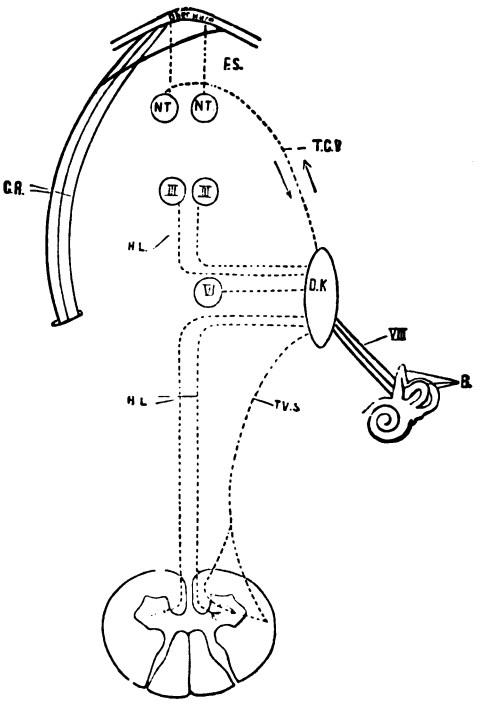

FIG. 389.—Diagram of some of the relations of the cerebellum to other portions of the nervous system. (After Bruce, taken from a paper by Bruns.) F.S. Sagittal fibres. N.T. Nucleus tegmenti. C.R. Restiform body. T.C.V. Tractus cerebello-vestibularis (et vestibulo-cerebellaris). III. Oculo-motor nuclei. VI. Abducens nucleus. VIII. Acoustic or vestibular nerve. B. Semicircular canal. D.K. Deiters' nucleus or vestibulbar nucleus. H.L. Posterior-longitudinal bundle. T.V.S. Vestibulo-spinal tract.

[1] A few writers (Wallenberg, Kohnstamm) regard these as having chiefly a cerebello-fugal conduction (see p. 641).

mechanism of the cerebellum. This interference is probably effected by the cortico-pontine tract which connects the frontal lobes with the opposite cerebellar hemisphere. It is well known that some writers localise the motor centres for the trunk muscles in the frontal lobes. Whilst these centres, as Bruns thinks, control the *voluntary* movements for the maintenance of equilibrium, the cerebellum exerts a reflex and automatic influence upon co-ordination, but these two central structures may act upon each other. Clarke and Horsley think the cortico-pontine tract arises mainly from the temporal lobe. We must finally refer to the important connections which the cerebellum maintains with the nuclei of the ocular nerves by means of Deiters' nucleus and the posterior longitudinal fasciculus.

Thus we can understand that disturbances of co-ordination—*cerebellar ataxia*—are chiefly produced by diseases of the cerebellum, but that they may also be caused by affections of the frontal lobes, the crus cerebelli, and all the tracts leading to and coming from the cerebellum. The diagnosis can in general be made only from the accessory symptoms, and from the intensity and the temporary appearance of the cerebellar ataxia. The anatomo-physiological conditions explain why the disturbances of co-ordination above described are produced mainly by affections of the vermiform process and the portions of the cerebellar hemisphere adjacent to it, and the fact that not merely *cerebellar*, but under certain conditions (lesion of the spino-cerebellar tracts) *motor ataxia* may also be caused by affections of the cerebellum, is thus comprehensible.

This conception would further explain the fact that a disease of the cerebellum which has developed gradually or involved only portions of it, may be to a certain degree masked by the cerebrum, as Anton (*C. f. N.*, 1903) has shown by a case, and as Sträussler has also described.

All those portions of the brain, lesion of which may give rise to ataxia resembling the cerebellar form, are anatomically connected with the cerebellum by tracts which pass through or arise in them.

The *diseases of the cerebellum* are very numerous and many of them have been described in other parts of this work. Hæmorrhages, softenings, inflammations, and very frequently abscesses and tumours (including cysts) occur in the cerebellum. It is not yet certain whether *toxic* diseases affect the cerebellum, but the effect of acute alcoholic intoxication and cases by Bechterew, Schnitzer, and others suggest that cerebellar ataxia may be produced by toxic influences.

I have in the earlier editions mentioned a case in which *hemicrania* manifested itself by attacks of a cerebellar type.

Pansini, and also Forli (*Bollet. Rom.*, 1907) describe a cerebellar syndrome in the pernicious form of malaria.

In all these conditions we must of course bear in mind that labyrinthine or vestibular affections may give rise to similar symptoms.

Atrophy and *sclerosis* of this organ has hitherto received little attention.

The earlier cases of this kind have been collected by Nothnagel and Hitzig, and to these numerous others have been recently added, amongst which I would mention those of Claus, Fraser, Sepilli, Hammarberg, Moeli, Nonne, Pierret, Menzel, Schultze, Arndt, Cramer, Spiller, Miura, Neuburger-Edinger, Lannois, and Paviot. Some of these, however, as Mingazzini notes, are cases of multiple sclerosis in which the process is located chiefly in the cerebellum, and to this class also belongs the case lately described by Schweiger (*Obersteiner*, xiii.). Dejerine and Thomas have made a very interesting contribution to the subject (*Nouv. Icon.*, 1900). Of the recent cases we should mention those of Michell Clarke, Rovese-Vecchi, Anton, Negele-Théohari, and in particular the exhaustive study of the question by Monsarrat-Warrington (*Br.*, 1902). They describe a combination of congenital cerebellar atrophy with spina bifida and other anomalies of development, and attempt to classify the published cases. We owe to Mingazzini (*M. f. P.*, xviii.) and Sträussler (*Z. f. Heilk.*, xxvii.) comprehensive studies which include a consideration of all the published cases. See also Voisin and Lépinay, "Syndromes cérébelleux congénitaux," *R. n.*, 1907; Steltzner, *M. f. P.*, xxiii.

Mingazzini distinguishes the following forms: 1. pure unilateral agenesis and atrophy of the cerebellum; 2. pure bilateral agenesis and atrophy of the cerebellum; 3. cerebellar atrophy associated with diseases of the cerebrum; 4. associated with diseases of the spinal cord.

In these cases we are dealing either with a *congenital* hypoplasia or a congenital malformation, in which some of the lobes—a whole hemisphere or even the whole cerebellum—are absent or but rudimentarily developed, or with *acquired* conditions which terminate in shrinking, hardening, and atrophy of the cerebellum or some portions of it.

Thus we have on the one hand developmental disturbances, and on the other localised lesions occurring before or after birth. Some of these are of vascular origin—inflammations, softenings, possibly hæmorrhages—and some apparently arise in the meninges.

The symptomatology depends upon whether the trouble has an acute or chronic onset, whether it is limited to one-half of the cerebellum or involves both hemispheres, and in particular upon whether the cerebellum is alone affected, or the lesion extends over the rest of the cerebrospinal nervous system.

From my own observations and from some of the pathological reports contained in the literature, I gather that there is a *cerebellar form of*

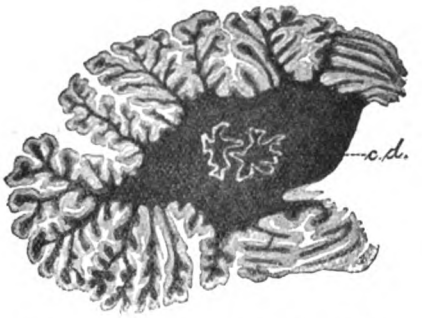

FIG. 390.—Normal cerebellar hemisphere. Transverse section. Pal's stain. *c. d.*, corpus dentatum.

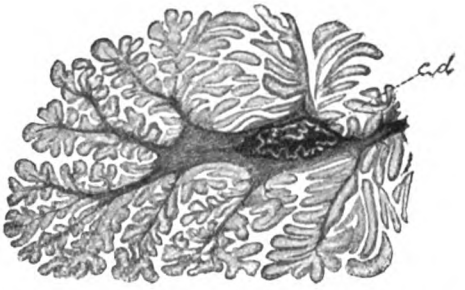

FIG. 391.—(Compare with Fig. 390.) Atrophy and sclerosis of the white matter of the cerebellum. Pal's stain.

infantile paralysis, in which lesions similar to those of infantile cerebral paralysis described on pp. 837 *et seq.*, may be found in the cerebellum. Anton has thus interpreted his case of " congenital absence of cerebellum with compensatory enlargement of other systems." In some of these cases the disease commences acutely with the picture of a severe brain disease, and subsequently develops into a condition of permanent paralysis. Thus it has often been found that children have been unable to walk in the first years after the onset of the disease, or that they could only crawl on all fours, whilst later they showed all the symptoms of cerebellar ataxia. The curable form of acute hæmorrhagic encephalitis may also be localised in the cerebellum (Oppenheim ; Nonne, see p. 828).

Batten (*Br.*, 1905) discusses these conditions in his comprehensive work upon the ataxia of childhood. Consult also the chapter on acute ataxia, p. 321.

In the case under the observation of Arndt and myself,[1] the disease was acquired in later life and was apparently due to atheroma. Here the process of sclerosis and atrophy was limited to the corpus dentatum (*cf.* Fig. 391).

The *symptoms* found in cases of cerebellar atrophy have not been of

[1] *A. f. P.*, xxvi.

the same kind in the various cases. In some, *e.g.* a case recently communicated by Wadsworth,[1] the defect in the cerebellum had produced no symptoms of any kind. The following group of symptoms is, however, usually present: *staggering gait* (*cerebellar ataxia*), *vertigo*, *and speech disturbance*, which is sometimes described as dysarthria and scanning, or intention tremor, sometimes as ataxia of the speech muscles. Exaggeration of the tendon reflexes (?) has been occasionally mentioned, and there have sometimes been *epileptiform attacks*, *paralysis of the ocular muscles*, or abnormal position of the eyes, and sometimes *tremor*, which is not sufficiently characterised. The mental development is often strikingly defective. According to the prevailing views above described, however, there can be no doubt that of these symptoms practically only the cerebellar ataxia and the vertigo, probably also the speech disturbance and the nystagmus, and possibly the tremor are dependent upon the lesion in the cerebellum itself. Apparently the corresponding symptoms of disseminated sclerosis may also be attributed to involvement of the

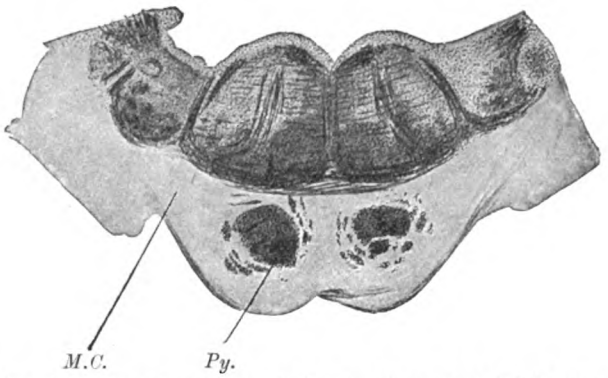

FIG. 392.—Atrophy of the fibres from the cerebellum to the pons and their processes in primary atrophy or sclerosis of the cerebellar hemispheres (case of Arndt-Oppenheim). *Py.* Pyramid. *M.C.* Fibres from the middle cerebellar peduncle (Weigert stain).

cerebellum and its peduncles (Babinski, Touche, Thomas). The hydrocephalus which is frequently found (Kirchhoff, Sommer) must be held responsible for the mental disturbances.

I have seen of late years a number of cases in which it was difficult or impossible to diagnose between cerebellar sclerosis and multiple sclerosis. The complete absence of any spastic and purely spinal phenomena were in favour of a cerebellar affection. The presence of these symptoms in doubtful cases decides in favour of multiple sclerosis, but we should not forget the existence of cerebello-spinal diseases of other kinds (see below). Thus acute disseminated encephalitis may extend to the cerebellum and spinal cord and produce corresponding symptoms, as I have several times observed. This disease is of course akin to mulitple sclerosis and may develop into it.

Further, cerebellar atrophy has been found in cases which show great similarity to Friedreich's disease (*q.v.*). Thus Nonne[2] describes a familial disease of which the cause was found to be an abnormal smallness

[1] See also Spiller-Robertson-Wadsworth: "Cerebellar Lesions without Cerebellar Symptoms," *Univ. of Penn.*, 1901. Also Saenger, Nonne. *N. C.*, 1907. I have along with Krause observed that deep incisions which divide the human cerebellar hemisphere from top to bottom do not necessarily give rise to any marked functional disturbances.

[2] *A. f. P.*, xxii., xxvii., xxxix.

of the whole central nervous system, especially of the cerebellum, the hypoplasia of which Nonne later regarded as being of decisive importance. This disease developed in three brothers at the period of puberty or in adult life, and had an absolutely chronic course. The symptoms were disturbance of speech (speech was excessively loud and explosive), nystagmus, imbecility, paralysis of the ocular muscles and simple optic atrophy, disturbances of co-ordination, and exaggeration of the tendon reflexes. It is obvious that all these symptoms cannot be attributed to the congenital smallness of the cerebellum.

Subsequently Marie,[1] referring to the cases of Nonne, Fraser, Saenger-Brown, and others already mentioned, described the syndrome of heredo-cerebellar ataxia discussed on p. 194, and ascribed it to hereditary atrophy of the cerebellum.

Other cases have been published which do not strictly belong either to one type or the other, and which have been interpreted as transition forms between Friedreich's disease and heredo-ataxia. These include among the older cases that of Menzel (*A. f. P.*, xxii.), and among the newer that of Sträussler (*Z. f. Heilk.*, xxvii.), and the conviction has more and more gained ground (Nonne, etc.) that no sharp dividing line can be drawn between these two forms, although there can be no doubt that congenital weakness or inferiority of the cerebellum and its processes plays an essential part even in the morbid conditions of this kind which develop in later life. This view is the more justified in that both these types of cerebello-spinal disease have been observed in the various members of one family (Nonne, Mingazzini).

The cases reported by Dejerine and Thomas are of great interest. They have had the opportunity of making a careful pathological examination of two cases which in their clinical symptoms were very closely allied to heredo-ataxia, and they found symmetrical atrophy of the cerebellum, especially of the hemispheres, which did not extend beyond the corpus dentatum, atrophy of the middle and of part of the inferior cerebellar peduncle, as well as of the grey nucleus of the pons Varolii, of the olives, accessory olives, arciform fibres, etc. The disease was chiefly distinguished from Marie's type by the absence of heredity and of the familial character. Pathologically the processes showed an extraordinary resemblance to the cases described by Arndt from my laboratory, except that the changes were not those of chronic inflammation but of pure atrophy. The writers regard the affection as a primary atrophic one—a kind of systemic disease—and they emphasise the fact that the picture drawn by Marie does not correspond to that of a uniform disease, but to that which may be produced by processes of various kinds in the cerebellum and its tracts.

See also Loew, *Thèse de Paris*, 1903; and as regards the finer histopathological relations of these cerebellar affections, Thomas (*R. n.*, 1905), Lannois-Paviot (*Nouv. Icon.*, xv.), and Sträussler (*N. C.*, 1906). A case observed by André-Thomas seems to show that the disease may be produced by structural changes which can only be detected by the microscope, the cerebellum having a normal appearance to the naked eye.

Thomas (*R. n.*, 1903) has also shown that there is a form which corresponds otherwise to the type described by Dejerine and Thomas, and in which the pyramidal tracts are involved, so that the symptoms still more resemble those of disseminated sclerosis. Clarke's case (*Br.*, 1902) is more complicated and ambiguous. In it the disease, apparently a familial one, manifested itself in a boy by the symptoms of heredo-ataxia and blindness, and there was found on post-mortem examination a sclerosis of the cerebellum and the occipital lobes. According to Bourneville-Crouzon (*R. n.*, 1904) there is a familial form of cerebellar atrophy which is associated with idiocy and spastic diplegia.

[1] *Semaine méd.*, 1893; see also Londe, "Hérédo-ataxie cérébelleuse," Paris, 1895.

A carefully studied case by Rossi (*Nouv. Icon.*, xx.) resembles those of Dejerine-Thomas, and still more those of Oppenheim-Arndt, but it is distinguished from the former by the fact that the olives and the grey nuclei of the pons Varolii were normal. Marie and Rossi describe as characteristic of this type the onset of the cerebellar disturbances of gait at the age of 60 to 70, the gradual progress of the disease, the possible appearance of slight spastic disturbances and of speech troubles and adiadococinesis. In Rossi's case and one described by Murri (*Riv. crit.*, 1900), the affection had a subacute development following a gastro-intestinal disease.

Frenkel and Langstein (*Jahrb. f. Kind.*, xi.) also describe a congenital familial hypoplasia of the cerebellum.

On the whole, therefore, much greater knowledge is required before we can draw clear and well-defined clinical pictures of the various diseases which mainly or exclusively affect the cerebellum.

THE NEUROSES

Hysteria [1]

The literature is for the most part to be found in the monographs of Gilles de la Tourette, "Traité clinique et théraр. de l'Hystérie," Paris, 1891 and 1895; Jolly, "Hysterie," in Ziemssen's "Handbuch," and also in Ebstein-Schwalbe's "Handbuch," 1900; Binswanger, "Die Hysterie," Nothnagel's "Handbuch," xii. The collected works of Charcot should specially be consulted.

Hysteria is a disease which has afforded the widest scope for errors in the conception and interpretation both of its various symptoms and of the disease as a whole. This striking fact is explained by the circumstance that the disease is one of the *mind*, which finds its manifestation less in intellectual disorders than in changes of character and mood, and which conceals its intimate nature behind an almost unlimited number of *physical* symptoms.

The disease chiefly affects women. Briquet's statement that the proportion is fifty men to a thousand women requires to be modified from later experience to about one in ten or one in six, or, according to Pitres and Gilles de la Tourette, with whose experience ours does not agree, to about one in two. The figures naturally cannot be quite exact.

The disease commences in *youth*, at the time of puberty and the years immediately following. It becomes evident very often at the end of the second and in the course of the third decade. Early childhood is by no means immune. I have not infrequently seen hysterical symptoms in children of 4 to 6 years old, and marked hysteria in children of 8 to 10, in boys hardly less often than in girls. Bruns,[2] Saenger,[3] Jolly, Ferrier, Bézy, and Bibent,[4] etc., have reported similar cases. I have had some remarkable cases in which the hysterical symptoms appeared in the second and third year (see chapter on eclampsia infantum). These have been lately confirmed by other writers, *e.g.* Bézy and Chaumier. It is very unusual for the disease to occur after the climateric. Senil hysteria has, however, been mentioned.

Although, so far as our knowledge goes, no people and no race is immune, the tendency to hysteria is by no means evenly distributed. It is specially common in France. Germans seem to be less susceptible, whilst the Jewish race furnish a singularly large proportion of such cases. The Poles seem also to have a special predisposition to it.

[1] Consult also the following chapter on neurasthenia and the appendix, as these neuroses are in many points allied and cannot be consistently separated from each other.

[2] "Die Hysterie im Kindesalter," 2nd edition, Halle, 1906.

[3] "Neurasthenie und Hyst. bei Kindern," Berlin, 1902.

[4] "Die Hyst. im kindl. und jugendl. Alter," German translation, Berlin, 1902. See also Oppenheim, "Die ersten Zeichen der Nervosität des Kindesalters," Berlin, 1904; B. Weill, *Thèse de Paris*, 1904; Eulenburg, "Mod. ärztl. Bibl.," 1905; Combe, "Die Nervosität der Kinder," Leipzig, 1903.

symptoms are specially apt to show this epidemic spread. School epidemics have been observed even in recent years, *e.g.* in Braunschweig.

The great danger of *spiritualistic* séances giving rise to hysteria and mental disorders has been pointed out by Charcot, Oppenheim, and Henneberg. Naturally it is mostly persons with a strong predisposition to this disease who give themselves to such pursuits.

All diseases which cause *loss of the fluids of the body* and *diminution in strength* may lead to hysteria. The relations between the uterus and this disease have been greatly over-estimated. Diseases of the sexual organs naturally tend specially to influence the psychical life. Women suffering from hysteria are often sterile and their sexual life is more or less abnormal; their marriage is usually unhappy, and this may be the source of the hysteria. The view that various affections of the genital organs, parametritis and perimetritis in particular, as well as retroflexion of the uterus, might cause reflex hysteria was very widespread among gynæcologists. It can usually, however, be discovered that the tendency to hysteria was manifest long before the onset of the uterine disease, and that the latter is merely an exciting factor. This point of view has been defended by Binswanger, although he attributed to diseases of the female genital organs the power of producing the most profound changes in the condition of the excitability of the central nervous system.[1] *Masturbation* should be mentioned among the etiological factors. Freud goes much too far, however, when he regards the "sexual trauma" of early childhood, accompanied by irritation of the genitals, as the specific cause of hysteria. I and others have seen hysteria frequently follow *castration*, but there was usually a pre-existing tendency to the disease. The troubles of the *climacteric* are partly due to hysteria and neurasthenia, and disturbances in the vaso-motor nervous system play a particularly important part in such cases (see section on diseases of the sympathetic).

Diseases of the gastro-intestinal system, of the nose and ear, may give rise to symptoms of hysteria, but only if there is pre-existing disposition. The nervous disorders caused by *intestinal worms* are partly hysterical in character.

Morbid conditions may be caused by chronic *alcoholism*, chronic *lead*, *bisulphide of carbon, and mercurial poisoning*, which are very much allied to hysteria, and which in Charcot's estimation are identical with it. Hysterical symptoms may also develop after chloroform and other narcoses. Acute infective diseases and syphilis are among the exciting causes of hysteria.

We do not regard the view that an *abnormal process of metabolism* is the cause and origin of hysteria as being based on any definite grounds.

Any *injury* which is associated with mental excitement may directly result in hysteria. On the other hand morbid conditions may follow injuries to the head, and general and localised physical concussion, in which hysteriform symptoms may play a prominent part (see chapter on traumatic neuroses).

[1] The modern conception of gynæcologists has been set forth in the works of Krönig, "Uber die Bedeutung der funkt. Nervenkr. für die Diagn." and "Ther. in d. Gynäk.," Leipzig. 1902; Theilhaber, "D. Zusammenh. von Nerv. mit Stör." in d. weibl. Geschl., Halle, 1902; Sutter, *M. f. Geburt.*, xxv., etc. The thesis of Freund ("Mod. ärztl. Bibl.," Berlin, 1904), whose views are akin to those of the old school, and of Hoenck ("Samml. zwangl. Abh.," 1905), should be referred to. E. Meyer (*D. m. W.*, 1906) has recently treated the question from the psychiatric point of view.

SYMPTOMATOLOGY

NATURE OF THE DISEASE. MENTAL CONDITION, ETC.

The definition, psychological analysis and nosological limitation of hysteria, are associated with very great difficulties, which lie partly in the nature of the affection itself and partly in the fact that it is often combined with other neuroses, especially neurasthenia, and with other psychopathic conditions.

The attempts to trace the nature of hysteria to a fundamental law, to compress it into a formula, have only succeeded as regards some of the symptoms, but have not led to any comprehensive definition.

We are only so far in a position to characterise certain fundamental phenomena and draw some psychological deductions, but we are not yet able to give an analysis of all the symptoms, forms, and cases.

Many writers have endeavoured to explain the nature of hysteria. In addition to the older writers (Sydenham, Briquet, Huchard, etc.), it has been specially studied by Charcot, Möbius, Janet, Breuer-Freud,[1] Vogt,[2] Loewenfeld, and Binswanger. The communication of my own [3] conception of its nature was published soon after the treatise in which Möbius set forth his theory, and the grounds upon which it was founded have already been expounded in earlier editions of this text-book.

Möbius, following Charcot's lead, held that all the morbid changes of the body caused by *imagination* are *hysterical*. I followed this by ascribing the greatest importance to the *exaggerated emotional excitability* and the morbidly *exaggerated influence* of mental emotion upon the otherwise normal motor, sensory, vaso-motor, and secretory functions. O. Vogt expressed a similar opinion. He regarded exaggerated emotivity as the congenital primary phenomenon of hysteria, and looked upon all its psychopathic symptoms as intensified changes of normal phenomena. Janet [4] is of opinion that it is "contraction of the field of consciousness" which makes it impossible for the hysteric to admit a number of impressions into his consciousness and to connect them with his own personality. He (and Sollier) think that unconscious and sub-conscious imagination plays an essential part in the production of hysterical phenomena. Breuer and Freud point out that mental trauma, which do not lead to an emotional discharge which relieves the mind, continue to a certain extent to exercise a latent effect upon it, to influence the moods, and to produce a *conversion of the emotional excitement into physical phenomena*, in such a way that these become independent of the original mental experience. This ingenious theory, which these writers advance with great clearness and which has been utilised in treatment, can merely be alluded to here. Vogt was able to demonstrate this process in another way. Krehl's [5] view is very much like our own, and is in accordance also with the teaching of Janet, Breuer, and Freud. Hellpach [6] thinks that the fundamental physiological symptom of hysteria is increase in the intensity, extent, and quality of the psychogenic and physical processes (the movements of expression in the widest sense of the word), and their disproportion to the movements of the mind. Binswanger agrees with us in emphasising the fact that sensations and ideas all exercise their emotional effects in an abnormally exaggerated way upon the cortical (and infracortical) functional mechanism, sometimes facilitating and sometimes inhibiting its action. The hysterical change consists in the fact that the regular correlation between the mental and organic series of cortical functions is disturbed in a double sense; on the one hand the mental process which corresponds to a certain number of organic cortical excitations is absent, and on the other an organic cortical excitation is associated with excessive mental activity, etc.

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[1] *Z. f. P.*, Bd. 1.; *W. kl. R.*, 1896 and 1898; *N. C.*, 1893-94; "Studien über Hysterie," Vienna, 1895; "Drei Abhandl. zur Sexualtheorie," etc., and *M. f. P.*, xxvii. 12.

[2] *Z. f. Hypnot.*, viii., etc.

[3] Oppenheim, "Tatsächl. und Hypoth. über d. Wes. d. Hysterie," *B. k. W.*, 1890.

[4] *Arch. de Neurol.*, 1893, and "Der Geisteszustand der Hysterischen." German translation, 1894.

[5] Volkmann's "Samml. kl. Vortr.," 1902.

[6] "Psychol. u. Nervenheilk., Philos. Studien," xix., and "Grundlinien einer Psychol. d. Hysterie," Leipzig, 1904.

Although the original teaching of Breuer and Freud must be admitted to be of great importance, its subsequent development and reconstruction by Freud has led him into a labyrinth, which we cannot too strongly warn the profession not to enter. The Zurich school—Bleuler, and specially Jung and Ricklin [1]—it is true, entirely agree with him, but many other writers, such as Hoche, Westphal, Aschaffenburg,[2] and Friedländer, strongly oppose his teaching. His attempts in particular to give a new interpretation to hysteria (and compulsive conditions), and his doctrine of its sexual origin imply an unwarrantable straining of the facts.

Among the other suggested definitions of hysteria we should mention that of Babinski, viz., that it is a mental condition which manifests itself by primary phenomena and secondary or accidental symptoms. The essence of the former consists in the fact that it may be produced in certain individuals by means of suggestion and dispelled by persuasion (" pithiatisme "). He regards the trophic disturbances as secondary. The artificial nature of this distinction need hardly be expounded. Raymond's [3] theory accords well with ours, and especially with Binswanger's.

We can merely refer to the discussion in the Paris Society of Neurology (*R. n.*, 1908), which took place during the publication of this work. It proceeds from an accepted definition of hysteria, and the defenders of this theory would exclude from hysteria all that does not fit into their definition. It is obvious that we cannot approve of this kind of criticism, which moreover, without any justification, entirely denies the psychogenic origin of vasomotor disturbances, etc. Raymond, Pitres, and O. Vogt, who was present at the discussion, have already expressed similar objections.

From all this we may make the following deductions :—

The primary cause of hysteria is an *abnormal condition of the mind*. The anomalies chiefly involve the *sphere of the emotions*; they consist in a lack of proportion between the intensity of the stimuli and the strength of the sensory reaction, usually in the sense that the emotional excitability is exaggerated, and the stimulus-threshold is diminished. Hence comes the inconsequence, the incalculable character of the reaction, one and the same stimulus leaving the mental sphere perhaps entirely unaffected, or giving rise to marked mental excitement and emotional displays of changeable quality. The disturbance is also made evident by the *duration* and *perseverance* of the mental excitement. On the one hand the feeling of discomfort awakened by an impression or an idea may persist for an abnormal length of time, and may produce a mood which is shown by its *pertinacity* to be morbid, and on the other the mental processes may be characterised by marked inconstancy and by abrupt, apparently motiveless changes. " Hysterical capriciousness " (moral ataxia, Huchard) is certainly not a constant sign, nor does it appear at every stage, but it is distinctly marked in the majority of cases. There may without doubt be also a pathological diminution of the mental reaction (" emotional torpor "), especially at certain stages of the illness.

One of my patients said that reading novels made her weep with emotion, whilst the death of her only and well-beloved daughter left her as impassive as if nothing had happened.

Another noteworthy factor is that past mental experiences " which have lost their intellectual substratum " may have a morbidly exaggerated influence upon the mood of the present, and that the reproduction of memory pictures which have unpleasant associations may be facilitated and increased.

[1] *Journ. f. Psychol.*, vii.; *M. m. W.*, 1906, etc.: Jung, *M. f. P.*, xxiii. See also Muthmann, Halle, 1907,

[2] *D. m. W.*, 1907, and " Verhandl. d. Amsterdamer Kongr.," 1907.

[3] " Nérvoses et Psychoses," Paris, 1907, and *Bull. méd.*, 1907.

Many hysterical symptoms are explained by the abnormal influence, upon which I first laid emphasis, of emotion upon the motor, sensory, vasomotor, and secretory functions, by the "disproportion" between the mental experiences and the psychophysical symptoms, or movements of expression which belong to them.

The connection of mental movements with these physical functions hardly requires to be insisted upon. Some writers (James, Lange, Sergi, Sherrington) even go so far as to regard the latter, the vasomotor, and visceral processes in particular, as the cause and origin of the feelings, *i.e.* they look upon the physical processes as primary, and ascribe the psychical processes to them. This is a standpoint which we can by no means adopt. In hysteria the proportion between these two series of symptoms is deranged, chiefly in the sense that the psycho-physical reactions are exaggerated. This characteristic is not peculiar to hysteria; it belongs also in a lesser degree to neurasthenia, and may occur as an independent isolated symptom in some definite region, *e.g.* an abnormal exaggeration of psychogenic vasomotor processes may appear in otherwise healthy individuals (due to heredity, intoxication, etc.). But in hysteria, *this exaggeration of the psycho-physical functions is associated with special and to certain degree pathognomonic features.* The movements of expression, although they may also be explained by a quantitative increase and retardation of normal phenomena, assume forms foreign to the normal life of the emotions, which do not even occur in neurasthenia. Thus spasms of laughing and weeping are symptoms of hysteria; the momentary inhibition of speech and movement from fright and anxiety may also amount to hysterical dumbness and paralysis; the indifference to painful stimulations which may appear normally during great mental excitement may amount to hysterical anæsthesia, the psychogenic vaso-motor processes may become hysterical cutaneous hæmorrhage, etc. In a similar way the feeling of discomfort caused by local irritation, whether due to trauma or local disease, may, as the result of morbidly exaggerated intensity and perseveration, become hysterical algia and hyperæsthesia after the irritating cause has disappeared.

Among the peculiarities of character in hysteria, we should include *exaggerated suggestibility.* Unfortunately the conception of suggestion is very vague and is apprehended very differently by different writers (Liebault, Loewenfeld, Vogt, Lipps, Syddis, Hellpach, Binswanger, Babinski, Bechterew, etc.).

It is clear that the idea associated with the feeling of expectation of an external or internal process creates on the one hand the condition of mind most favourable for its perception, but on the other hand it may mislead the judgment, so that indefinite sensations are mistaken for those which have been expected. The idea that a certain article of food may be over-salted or tainted may sharpen the sense of taste to such a degree that traces of a corresponding flavour, which would otherwise have passed unnoticed, will come to be perceived. A preconceived idea may, however, in normal persons awaken a sensation which is not produced by an adequate and real, but by the imagined stimulus. The idea that a fly has touched one's skin may arouse the sensation of itching, etc.

If we regard this mental process as *auto-suggestion* (in opposition to Hellpach, Babinski, etc., who emphasise the lack of sense and moderation in the content of the idea), we find that it is greatly exaggerated in

hysteria, and that it gives rise to new and abnormal phenomena by increasing the influence of the morbid imagination upon the sensations, and by greatly exaggerating the intensity of the physical processes of mental origin already described.

Thus the excitement associated with the idea that a blow or push has paralysed an extremity not only allows the pain to increase unchecked, but also engenders feelings of heaviness, etc., which exercise an inhibiting effect, so that the arm cannot be moved, etc.

If auto-suggestion is more effective in hysteria, so also is the influence of so-called foreign suggestion increased in a certain sense, but one must not confuse between suggestion and persuasion. The impressions coming from without and the idea awakened by encouragement may also influence the mental and psycho-physical processes in a higher degree in the hysteric than in the healthy individual, but it depends entirely upon the intensity and the character of the emotion created by the idea, and also upon the persistency of the contrasting idea present in the mind, whether the result of suggestion corresponds to the intention of the suggestor or has quite the opposite effect.

All this explains the fundamental quality of the hysterical symptoms, namely their instability. They may suddenly appear and as suddenly go, changes which may be almost entirely ascribed to mental influences. The attempt to replace the name hysteria by one corresponding to the sense (psychogenia, Sommer) is therefore justified.

The description of the *mental condition* of the hysteric is practically contained in what has just been said. The fundamental feature is the extreme *excitability*, but this does not react in the same way to all impressions; a morbidly exaggerated sensitiveness to certain stimuli is apt to be associated with indifference to others.

The patient's frame of mind undergoes abrupt changes, and may veer round without any recognisable cause. The remembrance of previous suffering is very vivid in the patient's mind, and creates feelings of discomfort which from time to time increase into outbreaks of pain or anger, and may give rise to conditions of dreamlike confusion in which he fails to recognise his actual surroundings and situation, and re-lives what is past or dreamed of as if it were all reality. Even events of his childhood may thus have a decisive effect upon the hysterical symptoms which become evident in adult life. The power to exercise *self-restraint*, to control and suppress the outward expressions of the emotions, is impaired.

Although the will power is usually affected in certain (physical) directions, we cannot as a rule speak of a general weakness of the will. The hysteric is apt rather to develop excessive energy whenever he desires to accomplish a definite purpose.

The actions may often seem to be irrational and motiveless, the reason being that the patient's morbidly exaggerated moods excite him to *explosive action*. To this may be also ascribed the inconsistency in the " character " of the hysteric. In spite of his marked egoism, we are often astonished by his peculiarities, which stand in glaring contradiction to his sayings and doings. The abrupt change of feelings will not permit of any stability of character; *the patient's mental life is not a unit*; he is composed of two or more personalities. As the hysteric is often m. judged by those around him, and unfortunately often by his physician also,

he becomes embittered, and often tries to represent his unappreciated sufferings in an exaggerated way, and thus shows the well-known symptoms even more distinctly and vividly than usual.

The uncommon cases in which hysterics have severely injured themselves and displayed their wounds as results of their disease (*e.g.* pressing live coals into the vulva) do not alone indicate the inborn tendency to deception and simulation, as has been wrongly thought, but point also to the severe disturbances of the mental condition which sometimes develop in the course of the disease. The hysterical lie is almost always pathological, and it may often be traced to a transformation of the memory pictures (pseudologia phantastica of Delbrück; mythomanie of Dupré; see *Bull. méd.*, 1905). Gottfried Keller gives an excellent example in his "Der grüne Heinrich."

As regards the results of the association test in hysterics, see Jung-Ricklin (*Journ. f. P.*, vii., and *Psych.-neurol.*, Woch., 1905).

The hysterical (or psychopathic) disposition may show itself early in the form of abnormal avarice.

It is surprising how little attention this phenomenon and its relation to psychopathic conditions has hitherto received.

The *mental power* is in general unimpaired. Hysteria is more often found in intelligent than in stupid persons; it may affect congenitally weak-minded and degenerate persons or be associated with conditions which lead to mental weakness, but such cases always represent merely a combination of independent affections. As a rule the *memory* does not show any general and persistent impairment. There is sometimes, however, great *inattentiveness*, *i.e.* the attention is so very much concentrated upon the inner processes or directed towards some special object, that external impressions are not perceived or fixed in the mind. Disturbances of association may be the result of the inhibiting influence of ideas which are associated with abnormally intense emotions. The reproduction of certain memory pictures may be rendered difficult because others remain fixed with pathological tenacity in the memory. Moreover, the attacks may leave blanks in the memory—*amnesiæ*—so that the remembrance of a certain period of time is entirely or partially wiped out ("retrograde amnesia"). The power of retaining new impressions in the mind is much less often impaired.

The blanks in the memory may relate to certain persons or events. The sentiments aroused by certain memory pictures (of relatives, etc.) may disappear for a longer or shorter interval of time and give place to complete apathy.

One of my patients stated that her affection for her husband and children only lasted so long as they were with her, and that each separation blotted it entirely out, although the memory pictures remained.

On the subject of amnesia, see Azam, Charcot, Garnier (*Bull. méd.*, 1905), Sidis-Goodhart ("Multiple Personality," London, 1905), etc.

Let us now consider more precisely the disturbances of the mental condition which come on in *attacks*; these include:—

1. *Conditions of Anxiety.*—Very many hysterical persons complain of an acute feeling of anxiety, which comes on in paroxysms and is associated with a feeling of pressure in the region of the heart or a sensation of palpitation; these may be the chief subjective troubles. The anxiety is usually not accompanied by definite ideas and fears.

2. *Hallucinatory Delirium.*—This is mostly a condition of dreamlike confusion, of sudden onset, in which the patient is under the influence of sensory hallucinations, usually of horrible visions, which he reveals by

his behaviour. The face has an expression of fear, horror, and anger; the patient turns round and round, flies as if from an enemy, crouches in a corner, etc., and seems to be quite oblivious of his actual surroundings. He can often be somewhat influenced, however; if he is approached he tries to escape or to strike out, or allows himself to be soothed for some moments. One can sometimes succeed in rousing him out of this condition by energetic opposition or by a strong cutaneous stimulus (sprinkling him with water). The memory of the attack and the content of the delirium is usually incomplete, but it is seldom altogether absent.

I have also seen attacks of a similar kind lasting usually only for some hours, and seldom for days, in hysterical children, and especially in *boys*; in one case the child was two years of age and of marked neuropathic heredity. This delirium may occur independently or as one of the symptoms of a spasmodic attack.

3. *Somnambulistic* and *hypnoidal* conditions (dazed conditions). On account of their close relationship to the spasms of hysteria they will be discussed along with these.

Marked mental disturbance of the character of melancholia, mania, or *folie raisonnante*, less frequently develop in the course of hysteria.[1] Although hysterical features my occur in this mental disturance and give to it a peculiar stamp, cases of this kind are not pure hysteria, but hysteria in combination with another psychosis. We can only, as Binswanger says, call it hysterical when it has developed out of obviously hysterical symptoms. The *fixed ideas* which occasionally develop in the course of the disease are not essential symptoms of hysteria, any more than are agoraphobia, dipsomania, and allied conditions. Their presence merely shows that the basis upon which the hysteria has developed is specially adapted for the development of other morbid mental conditions.

Disturbances of Sensibility.—Pain is never absent in hysteria. It may occupy any part of the body and assume the character of neuralgia, migraine, angina pectoris, rheumatism, or any other form of pain. *Headache* is very frequent. It does not always have definite features which reveal its hysterical nature. Some forms and accessory symptoms are, however, helpful in making the diagnosis. Thus the pain very often takes the form of *hyperæsthesia of the scalp*. The patient says that any slight touch of the scalp is painful and that he can hardly comb his hair on account of its sensitiveness (according to Head this peculiarity may be due to "referred headache"). *Clavus* is a well-known form of hysterical headache. This is very acute pain at a circumscribed part of the parietal or temporal region, which persists for hours and days and may be accompanied by symptoms of vertigo, nausea, or vomiting. Ordinary *hemicrania* is often associated with hysteria, and this form in particular has the tendency to develop from time to time into a permanent headache, persisting for weeks and even months (see hemicrania). *Occipital headache* is common; the pain is sometimes felt in the nape of the neck, sometimes in its neighbourhood, or it extends from the back into the occiput and forward into the region of the eyes. The headache, like all the symptoms of this disease, is influenced by the mind, and the subjective element is very marked in the description of it.

[1] As to these hysterical psychoses, among the more recent treatises those of Fürstner, "Die Deutsche Klinik am Eingang d. 20. Jahrhunderts," vi.; Raimann, "Die hysterischen Geistesstörungen, eine klinische Studie," 1904; and Raecke, *A.*; *P.*, Bd. xl.

The pain may be situated in the trigeminal region and be akin to neuralgia of the fifth nerve. The distinction of true from hysterical neuralgia has already been pointed out. Pain in the ear or mastoid process may develop after an ear disease.

Pain in the back is almost as common as headache. It sometimes affects the whole of the back, sometimes only certain segments, and is usually described as of a burning nature. Hemialgia, *i.e.* unilateral pain affecting all one side of the body, occasionally occurs.

Pain in the intercostal nerves, especially of the left side, corresponding to the type of an intercostal neuralgia, severe pain in the coccygeal region, associated with great sensitiveness to touch and with contraction of the muscles of this region (coccygodynia or sacrodynia), and pain which seems to be localised in the muscles, fasciæ, and periosteum, are very frequently complained of. There is also a hysterical form of *breast-pain* which has its seat in the mammæ (*mastodynia*). This may be very intractable, and may be combined with hyperæsthesia of the skin, redness, œdema, general and circumscribed swelling of the mammary glands, and even with "ulceration of the skin." This condition, described by Charcot and Gilles de la Tourette as "*sein hystérique*," as well as simple mastodynia, have been treated by amputation of the breasts.

Babinski and his followers seem to doubt the existence of this condition.

The so-called visceral neuralgias have very often a hysterical basis; there is an hysterical hepatalgia, nephralgia, cystalgia, etc. All these conditions have given rise to fruitless operations.

These pains are not due to local changes; they are of central origin and are to be regarded as pain-hallucinations (*psychalgias*), *i.e.* as direct stimuli of the pain-perceptive centres. It is true that they are often brought on by slight peripheral stimuli. The joint pains, arthralgias, and neuralgias of the joints, require to be specially considered. It is not unusual for the attacks of hysterical pain to occur only at certain times, *e.g.* in the night.

There are as many forms of *paræsthesia* as of pain. The sensation of formication and "pins and needles" is particularly common. *Hyperæsthesia* cannot be sharply distinguished from pain and is often combined with it. It is seldom complete, in the sense that every part of the body is abnormally tender to touch, which at any point elicits a feeling of pain. The hypersensitiveness is usually limited to *circumscribed* zones, to small patches of skin or corresponding areas of the deep parts (muscles, fasciæ, viscera), or it may occupy one extremity or the segment of a limb. Thus, the skin over a joint which is in the condition of contracture, may be hyperæsthetic. *Plantar hyperæsthesia* is also a symptom of hysteria.

The sensitiveness of the skin is specially felt in gentle stroking. In some cases the pain is only evoked by pressure penetrating the deep parts, the subcutaneous tissue, or muscles.

There is a form of hyperæsthesia that often occurs in childhood, in congenitally nervous and hysterical persons, in the nails (hyperæsthesia unguium, onychalgia nervosa) and hair. I have specially drawn attention to this condition (*M. f. P.*, xiii., and "Die ersten Zeichen der Nerv. d. Kind.," Berlin, 1904).

Although the mental factor plays the greater part in these hyperæsthesiæ, it is not always entirely responsible for them. I have sometimes been surprised to find that even minimal galvanic currents, opened and closed without the patient's knowledge, gave rise to marked sensations in the sensory nerves.

The hyperæsthetic zones are sometimes, though not always, identical with the so-called *hysterogenic zones*. These are circumscribed areas of skin, mucous membrane, or deeper parts, which form a focus of spontaneous pain; pressure upon these areas always elicits an attack (cramp, paroxysm of pain, etc.), preceded by an aura commencing in this area. In the same way pressure, usually strong, upon the same or other points (hystero-inhibitory zones) may sometimes arrest the attack. Among these hysterogenous zones, which may be situated at many different parts of the head, trunk (axillary line, the submammary and hypochondriac regions, etc.), in the testicles, the axis of the joints, etc. (Fig. 393), special consideration has been devoted to the region of the ovaries, because the peculiarities described are specially obvious at this site (Piorry, Schützenberger, Charcot). It is, however, very improbable that the ovary is the starting-point of the troubles, and that a hand introduced into the hypogastric region must necessarily touch this organ in order to elicit or arrest the attack. The corresponding zone usually lies higher than the ovary. Pressure on this spot may also be efficacious in some cases of male hysteria; indeed Steinhausen's[1] investigations show that this zone is very frequently sensitive to pressure in healthy men, so that its hysterical nature can only be inferred from an exaggerated degree of sensitiveness and the character of the reaction symptoms. The site of the apex-beat of the heart often forms a hysterogenous or hystero-inhibitory zone (Glozier). The stomach may also be the seat of hyperæsthesia. Hysterical pain in the back is usually associated with hyperæsthesia of the skin over the spinous processes of the whole spinal column or some part of it. It is acute, usually more so than the pain in the back which accompanies spinal diseases. The mental nature of this hyperæsthesia can usually be easily recognised from the fact that it does not appear if the attention is distracted.

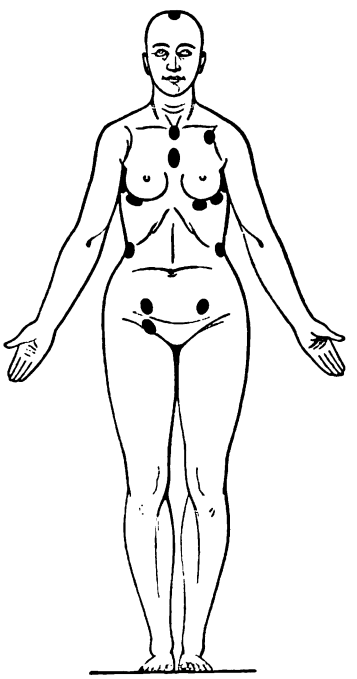


FIG. 393.—Hysterogenic zones on the anterior aspect of the body. (After Bourneville and Regnard.)

Hyperæsthesia sometimes also affects the *special sense organs*. Excessive sensitiveness to light and other feelings of discomfort in using the eyes, reading, etc. (*kopiopia hysterica*), marked acuteness of vision, and symptoms in the other special sense organs associated with certain *idiosyncrasies* (abnormal dislike to certain impressions of taste and smell, unusual preferences for others) and paræsthesiæ, *e.g.* swimming of objects before the eyes, noises in the ears, etc.—are some of the most common symptoms of hysteria. Optic hyperæsthesia may also be associated with an enlargement of the field of vision (Freund, Frankl-Hochwart and Topolanski[2]). The hyperæsthesia may take the

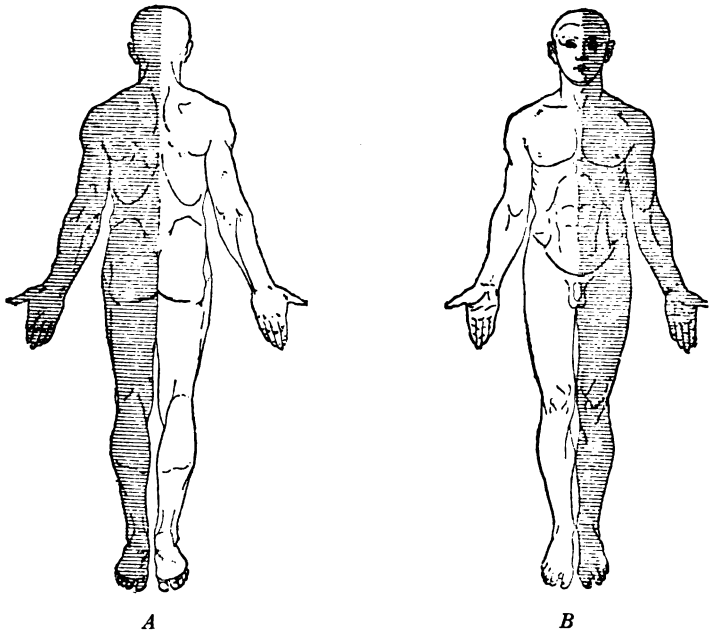
[1] *Z. f. N.*, xix.

[2] Deutschmann's "Beitr. z. Aug.," 1893.

form of *pathological after-sensations* (Binswanger), but this is very rare.

Anæsthesia is one of the most important diagnostic signs, as a diminution of sensation is found on objective examination in the majority or in a great number of cases (out of forty cases Pitres only twice found it entirely absent). Hysterical anæsthesia has a peculiar character on account (1) of its mode of distribution, (2) its close relation to disturbances of the sensory functions, and (3) of its mental basis and its reaction to external influences.

Careful investigations into this symptom have been made, in addition to the earlier writers (Gendrin, Briquet, Szolaksky), by Charcot, Pitres, Thomsen, and Oppenheim (*A. f. P.*, xv.),



A *B*

FIGS. 394 *A* and *B*.
Mode of distribution of hysterical hemianæsthesia. The shaded parts are anæsthetic. (After Charcot.)

Lichtwitz, Richter, Schmidt-Rimpler (*D. m. W.*, 1892), Wilbrand-Saenger ("Sehstör. bei funkt. Neur.," 1892, and *Jahrb. d. Hamb. Staats*, vi.; *M. f. P.*, i.), Frankl-Hochwart and Topolanski (*loc. cit.*), Reusz, Arnheim, etc. See also the bibliography in Schmidt-Rimpler, Nothnagel's "Handbuch," xxi., p. 312; and of the newer contributions, Klein (*A. f. P.*, Bd. xlii.).

We cannot accept Böttiger's view (*N. C.*, 1904) that all these disturbances are artificial and produced by suggestion, although it has been satisfactorily shown that they may be evoked, modified, and arrested by the influence of the mind. The view that hysterical anæsthesia is an artificial product (due to examinations, etc.), to which Bruns, Strümpell, and others had already referred, was specially defended in the discussion of the Paris Neurological Society. In any case these symptoms are much less common than they appeared to be from the original teaching of Charcot and our experience at the Charité.

As regards its *distribution*, anæsthesia is never confined to the areas innervated by certain nerves or nerve plexuses. It often assumes the form of hemianæsthesia, *i.e.* it affects one whole half of the body, more com-

monly the left, with the exception of the mucous membranes.[1] It ceases sharply in the middle line (Fig. 394), or less frequently just short of or slightly beyond that line. It less frequently extends over the whole surface of the body. In both cases we find as a rule circumscribed areas in which sensation is normal or exaggerated. The deep parts (periosteum, nerves, joints) are also anæsthetic. The loss of sensation is often limited to certain parts and segments of the body, and it then shows a peculiar kind of localisation, such as we do not observe in organic diseases of the nervous system (see Figs. 394-398).

A similar mode of distribution occurs in syringomyelia, but certain distinguishing points have been established (see corresponding chapter).

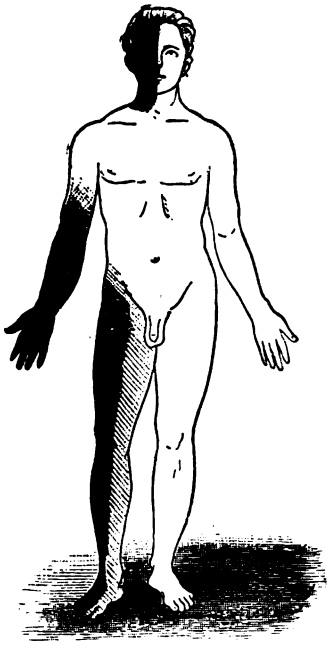


FIG. 395.

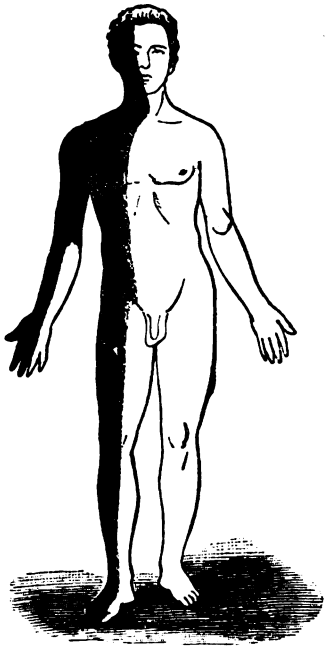


FIG. 396.

Mode of distribution of hysterical anæsthesia. (After Thomsen).

Thus it may be limited to the scalp below the hair and to the frontal region, forming a circle like a *cap* (Fig. 397). Or it may affect the arm and stop short at the shoulder-joint, involve the upper arm and the adjacent segment of the trunk in the form of a club or half waistcoat, or the hand and forearm in the form of a glove, etc. etc. As a rule it is bounded by lines which are drawn perpendicular to the longitudinal axis of the extremities ("amputation lines"). Thus parts of the body, which in the uninformed, popular idea form a unity (the arm, leg, hand, etc.), become insensitive. An insular distribution of the anæsthesia is much less common.

As regards the relation of the special sense disturbances, to the

[1] Binswanger recommends for the detection of the slight forms of anæsthesia the method of testing *bilateral symmetrical cutaneous* areas, which I had introduced as a method of clinical examination (compare p. 59).

changes in the sensory functions, anæsthesia of the skin and mucous membrane is usually associated with a reduction of function of the spinal senses. This relation is most markedly shown in so-called hysterical (sensory or mixed) hemianæsthesia. Here we find diminution or loss of sight, smell, hearing, and taste, on the side on which sensation is lost or impaired.

The *visual disturbance*, which is the most important of these symptoms, less often takes the form of diminution of the central acuity of vision than of *concentric narrowing of the field of vision*, which should be tested by the perimeter. The field of vision is equally reduced from every side, for

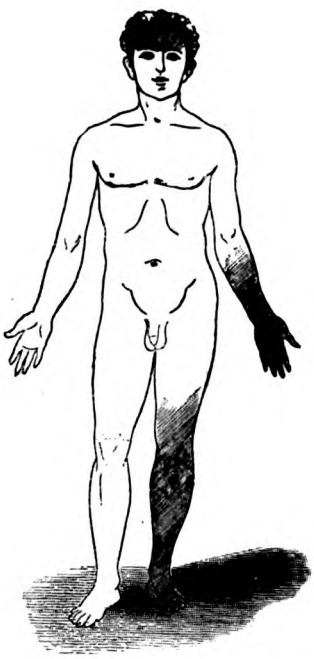


FIG. 397.—Type of distribution in a case of hysterical anæsthesia. The shaded parts are insensitive. (After Thomsen).

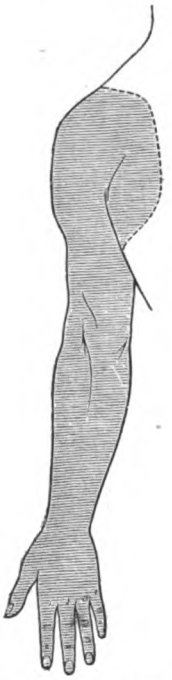


FIG. 398.—Mode of distribution of hysterical sensory disturbance. (After Charcot.)

white and colours in the typical order, except that occasionally the diminution for blue is more marked than that for red. *Dyschromatopsia* and *achromatopsia* are less common, *i.e.* the colours (especially blue and green) are confused centrally or are not recognised at all.

The term hemianæsthesia is not quite appropriate, as this narrowing of the field of vision is usually bilateral, but it is more marked on the eye of the anæsthetic side. There is seldom complete amaurosis of this eye, and cases of *total bilateral hysterical blindness* have very seldom been observed (see below). The diminution of the senses of smell and taste does not necessarily extend to all the qualities of these sensations. It may be that only certain portions of the mucous membrane by which taste is perceived, have lost their power of taste.

The nervous origin of the diminution in the power of hearing is shown by the reduction or absence of bone conduction, which is sometimes more accentuated for high than for low tones.

The functions of the special sense organs are not necessarily involved in any degree in the disturbance of sensibility; one or more of them may often be quite normal. In unilateral cutaneous anæsthesia, the special senses may be involved on both sides (this is the rule as regards sight), or even in rare cases they may be affected on the opposite side. The sensory disturbances are not invariably accompanied by anomalies of cutaneous sensation.

The anæsthesia may extend, always bilaterally, to the mucous membrane of the bladder and rectum, and cause secondary functional disturbances in these organs. Hemianæsthesia may also be found in the vagina (Briquet).

Character of the Anœsthesia.—It is not unusual for all kinds of sensation to be involved. In many cases there is simply *analgesia*, without impairment of the sense of touch, or *analgesia* and *thermo-anœsthesia*. Isolated thermo-anæsthesia is unusual. In the majority of cases there is merely partial diminution of sensation; instead of *anœsthesia* there is *hypœsthesia*, which may be so slight that it is only detected by comparison of the sensations evoked by stimuli of equal strengh applied to symmetrical parts of the two sides of the body.

The objection may be raised that a difference in the sensibility of the skin and sensory organs of the two sides, usually in favour of the left, is present under normal conditions (see Biervliet, ref. *R. n.*, 1898). This, however, is so slight that it is not brought out by our ordinary methods of testing. These objections and the corresponding data of Ioteyko-Stefanowska (*R. n.*, 1904), however, justify us in considering only marked differences as pathological. It has been sufficiently shown by ourselves and others that in such examinations one must as far as possible exclude the influence of suggestion.

It should further be remembered that analgesia to the prick of a pin does not always correspond to complete loss of the pain sense when the faradic brush is used.

In complete anæsthesia one may pierce a fold of skin or push the needle as far as the periosteum without producing any sensation. Nor does pinching the muscles or pressure upon the nerve trunks give rise to pain. The patient has no idea of the position of his limbs, and on this account he may be unable to carry out any movement when his eyes are shut. In such cases closing the eyes sometimes produces a condition resembling sleep (Strümpell [1]), which is probably of a hypnotic nature.

The name of *haphalgesia* (Pitres) has been given to the condition in which simple contact of the skin with objects such as metals, which normally produces merely a tactile or non-painful sensation, may elicit acute pain.

The relation of hysterical disturbances of sensation to the *reflexes* is not quite constant. The reflexes of the skin and mucous membranes are diminished or absent, corresponding to the hypæsthesia or anæsthesia, but complete loss of the corneal reflex is very rarely found. According to the experience of Liebrecht, Kempner, and ourselves, it is often diminished, but it should be remembered that even in healthy persons the intensity of this reflex varies within wide limits. The pupillary light

[1] *A. f. kl. M.*, Bd. xxii.

reflex (see below), the cremasteric, and usually also the abdominal reflexes are entirely unaffected. In cases of simple analgesia, the reflexes may react normally to tactile stimuli; when hypalgesia alone is present, they may be produced by strong painful stimuli.

The statements of H. Curschmann (*Therap. d. Geg.*, 1906.; *M. m. W.*, 1907) as to the condition of the reflexes and the blood pressure when painful stimuli are applied to the anæsthetic areas of skin in hysterics are practically a confirmation of my own observations (see section on traumatic neuroses). We should also refer to the interesting communications of Goldscheider on the psycho-reflex symptoms (*B. k. W.*, 1907).

It has been observed that cutaneous stimuli applied suddenly and unexpectedly may cause reflex movements, which do not occur when the patient is prepared beforehand for the test. This is probably in part due to the effect of fright. But as this phenomenon has sometimes been observed when sensation is apparently absolutely abolished, it proves the correctness of the view that hysterical patients do actually feel but are *not conscious* of doing so.

These facts are specially evident as regards the sensory disturbances. It has been proved by testing with the stereoscope or prism (Pitres, Westphal, Parinaud) that hysterics suffering from unilateral blindness can, at least in many cases, see with the eye which they think is blind. The power of orientation is not usually greatly affected, even although the field of vision is markedly reduced. In achromatopsia for certain colours it can sometimes be ascertained that the " mixed-colours " produced by blending these can still be recognised. All this shows that the patient does actually see, but that he is either not conscious that he sees or imagines that he does not do so.

Bilateral hysterical amaurosis is an uncommon symptom, but a great number of such cases have been recorded (Landouzy, Briquet, Dujardin-Beaumetz, Howship, Wecker, Mendel, Manz, Oppenheim, Abadie, Cramer, Kron, and others). According to H. Kron,[1] it should even be regarded as more frequent than unilateral amaurosis, but this conclusion is not justified by the cases published. In this amaurosis the pupillary light reflex is conserved, but it may be diminished and even for a time abolished by spasm of the sphincter pupillæ. In rare cases (Mauthner, Knies) the pupils are said to be dilated and rigid (?), but no case showed the symptom of pure isolated immobility to light. The condition, which tends to follow a spasm, usually lasts only for a short time, a couple of hours or days, or a few months (Briquet).

I have, however, observed a case of this kind in which the blindness lasted for months, then for years, and recurred thirteen times within ten years. The eyeballs were permanently in the position of convergence and could only be moved out of it partially and with difficulty; the upper eyelids drooped greatly (although there was no spasm of the orbicularis; see Fig. 399); the pupils were narrow, but reacted to light. The condition has now been stationary for many years. It has been exhaustively described by H. Kron, who has seen the patient in several of the attacks.

An intermittent form is also mentioned by Jüngken and Königstein.

It is said that persons suffering from hysterical blindness can find their way about better than other blind people, that they avoid obstacles, etc. In the case above mentioned I could not satisfy myself of the truth of this fact, and Crzellitzer also failed to find some of the attributes ascribed to hysterical amblyopia.

[1] *N. C.*, 1902. Consult the bibliography in this paper.

Unilateral hysterical *deafness* does not on the whole affect the hearing very much. The patient does not usually hold out the unaffected ear in order to catch the sound with it. In many cases he is unaware that he is at all deaf; it is even said that this unilateral difficulty in hearing or deafness is compensated by bilateral perception of sound. Bilateral deafness is mostly a very transient symptom. It follows mental excitement, an attack of spasm, or less often violent noise, in which the effect of the shock is the essential factor. The bone conduction is almost always lost in these conditions, and Rinne's test is quite negative. Hysterical deaf-mutism has occasionally been observed (Ballet, Mendel, Francotte, Vlis,[1] Schultze, Westphal [2]). Subjective noises in the ears do not always correspond to hysterical deafness.

In an interesting case of hysterical deafness which I observed along with Treitel, the condition developed after mental excitement in a little girl who had inherited the tendency, her father suffering from a nervous disease of the ear with hypochondria. The first attack lasted ten days,

FIG. 399.—Bilateral hysterical amaurosis with hysterical ptosis.

FIG. 400.—Condition of the frontales in true ptosis. (Compare with Fig. 399.)

then suddenly disappeared and recurred fourteen days later, etc. In the third attack there was aphasia for nouns. I was specially struck with the absence of any attempt to apprehend what was said to her or to read from the lips. In a patient of A. Westphal's this was not the case, but the condition was modified here by the fact that the patient had already associated with deaf persons in an institution. F. Schultze (*D. m. W.*, 1901) observed a boy suffering from hysterical deafness unconsciously continue a melody whistled near him. Barth (*D. m. W.*, 1900) also noted that the musical perception was unaffected in a case of absolute bilateral deafness. The combination of deafness, mutism, and blindness has been described by Marinesco (*Gaz. des hôp.*, 1899). Chavanne has discussed in detail the hysterical auditory symptoms ("Oreille et Hystérie," Paris, 1901).

Hysterical *anosmia* is as a rule merely one of the symptoms of sensory anæsthesia, but it may occur independently. In one of our cases it was present from birth in a mother and daughter, and was therefore to be looked upon as a stigma of degeneration.

The sensory disturbances are often associated with cutaneous and mucous anæsthesia of the corresponding openings of entrance into the sensory organs (external auditory meatus, etc.).

Patients suffering from hemianæsthesia and other sensory disturbances have often no suspicion of this defective sensation. They notice that

[1] *M. m. W.*, 1899.

[2] *Charité-Annalen*, xxiv.

something is out of order, and not infrequently complain of pain in some region of the affected side, *e.g.* unilateral headache, which radiates towards the arm and shoulder, but the loss of sensation is only revealed by medical examination. The anæsthesia may be associated with hyperæsthesia: an area of skin on which painful stimuli are not perceived as painful may be hypersensitive to the slightest touch, which may elicit acute pain.

The reverse symptom of a combination of tactile anæsthesia with hyperalgesia at the nipples has been reported by Graves (*Journ. Nerv. and Ment. Dis.*, 1905) as a stigma of degeneration.

Hysterical disturbances of sensibility are very variable. They may suddenly come and as suddenly go, after an attack of spasm, some mental excitement, or some slight injury, but in some cases they are very persistent.

It is an interesting fact that in many cases the anæsthesia has been successfully influenced by many kinds of manipulations, which cause it either to disappear or to change its position. This is specially the case as regards hemianæsthesia, which may disappear after even one application of the faradic brush. The anæsthesia may sometimes be transferred to the other side by the application of a metal or a magnet. The disturbances of the special senses may also pass in this way over to the other side. It may even be possible to effect the transfer by laying the metal on the skin of the sensitive side. The statement that different individuals respond to different metals has not been corroborated, and no credit can be given to the assertion that the metal, given internally, will work a cure. The transfer by the use of sinapisms, the laying on of wood, bone, and true and false magnets is also reported. It is very probable that these exercise a purely mental influence, and that the transfer is merely the result of suggestion. Some writers think that the true magnet exerts a specific influence upon hysterics (? ?). Féré (*Rev. de Méd.*, 1902) has again maintained that the magnet has an effect upon man, but this is a daring and unproved hypothesis. The anæsthesia is sometimes not transferred by this method, but is simply dispelled, or made to disappear after passing several times from one side to the other. In some cases the transfer takes place very rapidly; in others it may be half an hour and more before the effect becomes apparent.

Disturbances of motility, symptoms of irritation, and spasms are symptoms of hysteria which are absent only in a few cases. These forms of spasm are very numerous. *Emotional* and *respiratory spasms* are peculiarly typical. Laughing and weeping may be so exaggerated as to become convulsive. The morbid nature of these paroxysms is shown by the intensity and duration of the emotional manifestation, and the inability of the patient to *control* it. The respiratory muscles may be affected independently of the emotional spasm. Attacks of violent acceleration of the breathing (up to 180 per minute) associated with anxiety and oppression are a very common form of spasm. In one case they were elicited by any noise. In these conditions of tachypnœa objective signs of want of air are hardly ever present. Moreover, they almost always disappear during sleep (Dejerine). Hofbauer[1] gives remarkable data as regards differential diagnosis. Compare also Loubry.[2] Raymond-Janet attribute a type of respiration allied to the Cheyne-Stokes to an hysterical basis. Clonic spasms in the abdominal muscles, which last for days, hours, and even weeks, are an unusual form.

Singultus (hiccough)[3] and *eructations*, which are generally due to swallowing air (*aérophagie hystérique*), and which occur not only in hysteria

[1] "Semiol. und Different. der verschied. Arten von Kurzatmigkeit auf Grund der Atemkurven," Jena, 1904.

[2] *Thèse de Paris*, 1906.

[3] An epidemic onset of this has occasionally been observed, and was recently described by Abeles.

and neurasthenia, but also as a tic (Bouveret, Perrody,[1] etc.), and as an independent symptom arising from the psychopathic diathesis, paroxysms of *yawning* and *sneezing* (these respiratory phenomena being constantly repeated, possibly for hours), may also occur. I have treated hysterical individuals who had eructations whenever the body or certain parts, *e.g.* the region of the eyes, were touched. *Spasm of the glottis*, viz. attacks of spasm affecting the laryngeal muscles which contract the glottis, accompanied with severe sensations of suffocation, is a much less common condition. It is not entirely without danger, and has even on occasion called for tracheotomy. This spasm also occurs when the patient attempts to phonate: *aphonia spastica* (Schnitzler). There is a non-hysterical form of this disease (Semon). *Tussis hysterica* is a violent, usually hoarse, dry cough, resembling barking or other animal noises. The fits of coughing often return in a very regular, rhythmical manner. The individual attack may last several hours. Simple coughing, which has given rise to errors of diagnosis, may also be a symptom of hysteria. It is important to note that hysterical coughing almost always disappears during the night. *Blepharoclonus* and *blepharospasm*, which are often associated with photophobia and lachrymation, are not uncommon forms of spasm.

Clonic spasm of the diaphragm seldom occurs. Fox describes one such case in which the condition persisted for a year, and Barth reports an asthma phrenicum due to clonic spasm of the diaphragm.

Torticollis may also appear on the basis of hysteria.

This view is, however, opposed by S. Knapp, *A. f. P.*, Bd. xxxix., Broca-Herbinet, *Nour. Icon.*, xviii., and Kollarits, *Z. f. N.*, xxix. See also the section on tic.

It is not yet certain whether *globus*, one of the most common troubles in hysteria, is to be regarded as a spasmodic condition or as a paræsthesia. It is characterised by a sensation of pressure in the throat or larynx, as if a ball were firmly settled there, or the patient feels as if a ball were pressing up from the stomach or the abdomen into the throat. Buch[2] has studied this matter in detail. He regards globus as a symptom arising from the sympathetic, associated with sensitiveness of this nerve to pressure, and often combined wtih other signs of involvement of the sympathetic. If this be so, his statement that globus is not ultimately connected with hysteria must be altogether rejected. It is not probable that this symptom is due to local spasms in the muscles of the pharynx or œsophagus, but there is a form of hysterical *dysphagia* which is due to spasmodic closing of the œsophagus.

Cases of this kind have been recently reported by Thiercélin, Rosenheim, Schmidt, Eliot, Russell, Fürst, Rossolimo, Hartenberg, Bregman, Philippi, Levinsohn, etc. The suddenness with which the difficulty in swallowing appeared and disappeared was characteristic in some of these cases. In one (Fürst) the spasm came on when the tube was introduced, and it could only be removed during narcosis. As regards spasm of the pylorus and its occurrence in childhood, see Heubner, *Therap. d. Gegenw.*, 1906. The dysphagia is more often due to a phobia or fixed idea, and is then to be regarded as a direct symptom of hysteria. A local spasm of the intestinal muscles undoubtedly occurs in hysteria and may give rise to almost all the symptoms of intestinal obstruction and appendicitis (Spencer Wells, Bamberger, Briquet, Leube, Cousot, Nothnagel, Strauss, Schloffer, etc.). This condition has repeatedly been treated by laparatomy. Sander has published interesting cases of this kind and special attention should be drawn to Kausch's collection of his own and other cases (*Mitt. aus d. Grenzgeb.*, xvii.). He points out that in the majority of cases

[1] *Thèse de Paris*, 1901

[2] *A. f. P.*, Bd. xl.

the general condition did not correspond with the severity of the abdominal symptoms, that the disease might last for days, months, and years, that laparotomy reveals either no change at all or merely a contraction of a loop of intestine. Hysterical constipation may also be due to spasm of the intestinal walls.

In many cases of hysteria there is a tendency to painful conditions of spasm in the organs with smooth muscles. I have found this to be specially marked in some patients who had for years taken part in spiritualistic séances, and it seems to me that the kind of introspection thus engendered is very apt to produce an increasingly intimate connection between mental and physical processes in these systems. There may also be severe cardiac symptoms.

I have observed peculiar tonic spasms of short duration in the tunica dartos in a man who was apparently not hysterical.

Any of the muscles under voluntary control may be attacked by hysterical spasms. In many cases there are tremors which involve one or more extremities and produce *rhythmically* repeated *stereotyped movements*; in others the limbs are flung backwards and forwards in wild disorder. The movements are always such as might be voluntarily executed, but in hysteria they are produced with a force and persistence which would only be possible to the healthy individual if he exerted all his strength,

FIG. 401.—(After De la Tourette.) Phase of hysterical attack ("arc de cercle").

and which even then he could not keep up for many hours. In these localised spasms, the patient is as a rule quite conscious, but his mind may be obscured by *sensory hallucinations* or *morbid ideas*.

In order to make the nature of *general hysterical spasms* clear, we shall describe it in its severe form of attacks of *grande hystérie* (Charcot).

It should not of course be forgotten that Charcot's description was founded upon cases which had passed through the school of the Salpêtrière, and had been artificially cultivated and further developed by unconscious suggestion as well as imitation and mental infection. The cases to which Charcot's description of major hysteria apply have, as Raymond admits, become much more rare even in the Salpêtrière.

In fully developed cases, the attack may be divided into several phases, which show a certain regularity in the order of their occurrence and in the character of their symptoms. *Prodromata* in the form of depression, exaggerated excitability, a feeling of anxiety, palpitations, globus, etc., are usually present. These are followed by an *aura*, consisting of a sensation of a ball rising from the stomach or ovarian region into the throat, associated with great *anxiety*, palpitations, noises in the ears, a mist before the eyes, and impairment of consciousness. Then comes the *epileptoid period*, a spasmodic condition which has a great resemblance to the first

stage of the epileptic fit. The eyes close, the patient falls to the ground (not as a rule so suddenly as in epilepsy and without injuring himself), the head is drawn backwards or to the side, the jaw muscles are firmly contracted, or, if the trismus is incomplete, the tongue is protruded, breathing is arrested, the face becomes red, then cyanosed, the arms are extended or adducted to the trunk, the hands clenched, and the legs extended at every joint. This stage of tonic rigidity, which is usually of short duration, is followed by *clonic spasms*, in which the cyanosis disappears, the breathing returns but is generally accelerated and very loud, and the pupils, which are usually at first contracted, dilate. These clonic spasms introduce another phase of the attack, viz., that of *contortions* and *wide movements* (clownism). The face is grotesquely distorted, the extremities thrown about wildly and constantly changed into all kinds of positions. The patient clenches his fists as if in rage, flings his limbs widely about, throws his legs into the air, crosses them, bows the upper part of the body forwards, bends the head far back, and describes such a curve with the trunk that only the head and the heels rest upon the ground (*arc de cercle*) (Figs. 401, 402), rolls the body round its axis,

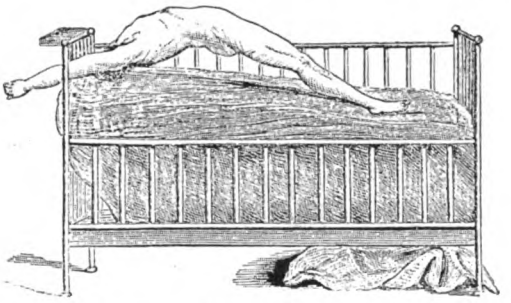


FIG. 402.—Phase of hysterical spasm. (After Charcot.)

or throws it from one side to the other, flings himself against the wall, etc. This muscular frenzy may be accompanied by weeping, screaming, howling, cursing and swearing, raging, or by spasms of laughing and weeping.

Next follows the period of *plastic, passionate gestures*. Whilst the mind is under the influence of sensory hallucinations and is passing through bygone or imaginary situations, the expression of the face and the postures of the body conform to the mental attitude. Fright and shock, anger, rage or rapture, suspicion and ecstasy may be expressed in the face or the attitude of the body with a fidelity of reproduction that might awaken the envy of an actor. The sensory hallucinations may often be influenced by external stimuli. The next phase is that of *more subdued delirium* in which *animal hallucinations* usually play a part. The attack gradually passes off and often leaves behind it disturbances of sensation, of movement, and other anomalies, but deep stertorous sleep hardly ever occurs in the post-paroxysmal period. It may be followed by a lethargic condition, not usually associated with general muscular relaxation, but with general or partial tonic contractions of the muscles (Binswanger). The individual attacks may last for a quarter to half an hour and more. It is not uncommon, however, for a number of attacks to follow

each other in unbroken succession and to constitute a *status hystericus*, lasting for one or more days. In this, as opposed to status epilepticus, the temperature remains normal.

Incompletely developed attacks are much more common than these major attacks. They consist of one or more phases of the latter, and show every conceivable combination of the symptoms. Thus they may be limited to the aura and the first epileptoid stage, and so closely resemble an epileptic attack that only careful examination during the attack can definitely decide between the two conditions. In the hysterical attack the movements are usually more excessive and indicate to some extent that they are under the *control of the will*. Further, consciousness is hardly ever entirely lost, and the signs of absolute unconsciousness, in particular the loss of the pupillary light reflex, are very seldom found (see, however, further on, and the chapter on epilepsy). The attack may also sometimes be influenced by external excitements, the contractions becoming more violent if one approaches the patient and tries to calm him; or some transient facial expression of emotion, an "arc de cercle" described by the body, or some other peculiar feature will reveal the nature of the attack. The tongue is never bitten, although the patient sometimes bites his lips. The *arresting effect of pressure upon the ovaries* during the attack may be often of value as a diagnostic sign.

FIG. 403.—(After de la Tourette.) Phase of the hysterical attack ("attitude passionelle").

A combination of hallucinatory delirium with clonic muscular or respiratory spasms may represent the attack. The emotional and respiratory spasms mentioned above (and spasm of the glottis) may be associated with features of a major attack.

The hysterical spasm sometimes corresponds to the type of *Jacksonian epilepsy*, *i.e.* it is limited to one side of the body, commences in certain muscles, and spreads to the others. The patient is usually confused but not unconscious. These attacks can generally be elicited or arrested from the pressure points or by some mental influence. During them the temperature is never raised. The attack may be limited for a long time to one extremity before it becomes general.

I have often observed this form, but as a rule have easily detected its hysterical character. See also Ballet-Crespin, Loewenfeld, Binswanger, Woltär (*Prag. m. W.*, 1904).

Finally, we should point out that attacks occur in hysteria which are allied to *petit mal*. I have seen undoubted cases of this kind. Binswanger describes syncopal attacks which resemble simple fainting (often with fluttering of the eyelids).

The memory defects or amnesia already mentioned (Azam, Charcot, Souques, Boris, Sidis, Garnier, etc.) are specially apt to follow repeated hysterical attacks. The amnesia is either partial or total. The cause

of the amnesia, or the events that preceded it are very often lost to the memory. The amnesia may refer to a definite group of memory pictures, to a certain person, etc. The power of apprehending and fixing new impressions is less often lost, so that an amnesia regarding events which happen subsequently to the attack (amnésie rétroantérograde) is less common.

Hypnoid Hysterical Conditions.—A number of hypnotic symptoms which have been termed various stages of the hypnotic condition, and may be articificially produced and blend with each other, may arise spontaneously in hysteria. These include *catalepsy*, *lethargy*, or *hysterical sleep* and *somnambulism*.

Catalepsy usually develops suddenly after some mental excitement, but may also recur periodically without this cause. Its most characteristic symptom is a condition of rigidity of the extremities, which when passively moved remain persistently for hours in the position into which they are put, and to any attempt to move them *offer slight resistance*, as if they were made of wax (flexibilitas cerea). This rigidity is either general or limited at first to certain limbs. It may attack the patient so suddenly that the body becomes fixed in any position. Respiration and the action of the heart are weakened and may be retarded. Sensibility is abolished, and so are the reflexes; the corneal reflex, however, is almost always conserved. The patient is usually quite conscious, or nearly so; at least many patients say they have heard what was being said by those around them but have been unable to speak or move. There is more often a dreamy condition, the patient being under the influence of sensory hallucinations. The eyes are mostly closed; if they are open the gaze is fixed and the face expressionless. The patient can often be brought out of this condition by stimulating the skin (douches, faradic brush), but it may last for days, weeks, and even months. Incomplete attacks of catalepsy, in which the rigidity spares some of the limbs, may also occur. Further, the rigidity is not always waxlike; in many cases the limbs offer the greatest resistance to attempts at passive movements. Catalepsy either appears independently or is associated with attacks of hysterical spasm.

No little interest is attached to the hysterical stupor or *lethargy*, described specially by Briquet, Charcot, Richet, Ballet, Lamacq, Pitres, Gilles de la Tourette, and Loewenfeld.[1] It is often related to attacks of spasm, but it may be an independent symptom. Headache may occur as a prodroma, but the sleep usually comes on suddenly, though it may be preceded by a period of drowsiness and apathy. On superficial observation the patient looks as if he were in a *deep sleep*. The muscles are not, however, always fully relaxed; indeed they may show all the transitions between slight tension and fully developed contracture. The masseter muscles in particular are often contracted. The eyelids are sometimes seen to flutter. The face is usually normal in colour and fulness, but it may be pale and livid. The breathing and the action of the heart may be much retarded, and the *respiration may even stop* for some minutes (hysterical suspended animation). In a case of Pfendler's (quoted by Binswanger) no sign of life could be detected for forty-eight hours, and everything was prepared for the interment. Interesting cases have recently been reported by Marestant, Beckers, and Rosenthal.

[1] *A. f. P.*, Bd. xxii. Of the newer works see Raecke, *B. k. W.*, 1904.

According to Charcot, Bourneville, and de la Tourette, the central temperature of the body is somewhat increased (up to 1°), a very valuable criterion for the differential diagnosis. The condition of the sensibility varies, but in contrast to natural sleep there is usually complete anæsthesia to mechanical stimuli. On the other hand stimulation of hyperæsthetic zones may sometimes provoke defensive movements, and may often even interrupt the attack. Movements also occur during the attack which give one the impression of being voluntary or reflex. The reflexes, even the corneal, may be abolished, although the tendon reflexes are always conserved (see below, however). Food put into the mouth is usually swallowed, but the deglutition reflex may be absent and feeding may be a matter of great difficulty. The patient wakens suddenly or gradually out of his sleep, and usually shows complete *amnesia* for the time it has lasted. On the other hand some patients state that they have been conscious of all that has happened during the attack, and have even felt the cutaneous stimuli. These attacks last for varying periods, usually only for some hours, but sometimes for many days, weeks, or months (Charcot, Bourneville, Gairdner, Krauss, Jolly), for one and a half years (Pfendler), and in a case of Lancereaux's [1] for twenty years.

See also the interesting data with regard to Gesine Meyer, who slept for seventeen years (*B. k. Q.*, 1904-05), and Eulenberg (*Mcd. Kl.*, 1906). There is no doubt, however, that cases are described as hysterical which should be regarded as belonging to dementia præcox or to a condition which is still unexplained. Thus it is very doubtful if Grober (*Z. f. N.*, Bd. xxviii.) was justified in calling his case hysteria.

Under the name of *narcolepsy*, Gélineau (*Gaz. des hôp.*, 1880), Westphal (*A. f. P.*, vii.), and others have described attacks of sleep of short duration and sudden onset. The patient who does not react to sound may sometimes be roused by a light touch. Friedmann (*Z. f. N.*, xxx.), who discusses this and allied conditions, points out that this is not real sleep, but a kind of transient *mental rigidity*, accompanied merely with a degree of confusion and loss of muscular power. The attacks may last from a half to several minutes They are often brought on by laughing (Gélineau, Loewenfeld). Narcolepsy certainly occurs in other diseases, especially as the equivalent of an epileptic attack (?), and from mental degeneration, but it is often of an hysterical nature. It may also occur in general obesity (Gélineau, Sainton, etc.), but this condition is in our experience often associated with hysterical and neurasthenic symptoms. Dosing accompanied by vivid day-dreams may also be symptoms of hysteria.

Somnambulism is either related to an attack of major hysteria forming to a certain extent a prolonged third stage of it, or it appears independently in the form of hallucinatory delirium. The hallucinations and illusions of the patient may often be influenced by sensory stimuli. In these conditions he may carry out complicated actions and display considerable acuteness of sensory perception. During the attack the memory for what occurred during previous seizures is conserved, whilst in the free intervals all that has taken place during the somnambulistic condition is as a rule forgotten. The whole condition and demeanour of the patient usually shows that he is under the influence of some abnormal mental state, which absolutely cuts him off from the external world. He less often appears in his "*second nature*," which may last for weeks and months, not greatly altered in his external appearance ; it is only his character and disposition which seem to be transformed. These are the cases in which one may speak of a *doubling of the personality*, or of the twofold

[1] *Acad. de Méd.*, 1904 ; *R. n.*, 1904.

nature of the mental individuality.[1] The *hysterical crepuscular state*, in which the patient behaves in a very childish way, giving absurd, senseless answers to simple questions, etc., and at first making the impression upon one that he is deceiving, is a variety of these attacks (Ganser,[2] Binswanger,[3] A. Westphal,[4] Raecke,[5] Vorster,[6] Henneberg[7]). Nissl's view, that these cases represent a form of catatonic negativism, is disputed by other writers; but it must be admitted that the symptom of senseless talking may also occur in other psychoses. Moreover, a condition may develop in which the patient imagines himself back in an earlier period of his life, especially in childhood, and speaks and acts accordingly. This symptom is known as *ecmnesia* (Pitha, Fontenille, Krafft-Ebing).

Somnambulism (sleep-walking) is also the name given to similiar conditions in childhood and youth in which the patient leaves his bed during the night, wanders round in his sleep, and carries out complicated actions, which he has entirely forgotten when he wakens. These conditions may be of a hysterical or epileptic nature, or may be due to general mental deterioration, although they may not precisely correspond to any of these types. Charcot was of opinion that this somnambulism is seldom of a hysterical nature, but this is by no means Dejerine's experience or my own. Mesnet defines noctambulism as a dream translated into speech and action, and therefore as a pathological exaggeration of the dream process.

This class includes further those peculiar conditions of the wandering mania (*Wandertrieb, automatisme ambulatoire*, fugues, dromomania, poriomania,[8] etc.), in which the patient, apparently without any motive but under the mastery of some internal influence, leaves his home, walks long distances, or undertakes long journeys. He appears outwardly unchanged, but his mind is morbidly affected, dreamily altered, and limited, so that on waking out of this condition he has merely an incomplete, dream-like memory, or rarely a complete amnesia for what has happened during this period. It has been found that the remembrance of the experiences through which the patient then passed has returned when he was hypnotised (Tissié). It was formerly thought that ambulatory automatism was a symptom of epilepsy, and some writers, such as Aschaffenburg and Schultze,[9] still express a somewhat similar opinion. There is no doubt, however, that it frequently has a hysterical basis (Raymond, Bregmann, Jolly, Oppenheim). Heilbronner[10] regards hysteria as the most frequent cause. Dejerine speaks of a neurasthenic form of this symptom. I have treated a case in which these attacks alternated with hysterical convulsions. Hysterical and neurasthenic conditions of this kind lack the violent, unrestrained character, the profound impairment of consciousness and the total amnesia. According to Heilbronner these attacks are mostly the morbid reaction of degenerated individuals to a condition of ill health. Although serious crimes are seldom committed during these attacks of hysteria, the patient may come into conflict with the law and doubt may arise as to his responsibility. The difficulties which may occur in deter-

[1] Compare on this matter the recent paper by Sidis-Goodhart, "Multiple Personality," London, 1905; ref. *Journ. f. P.*, vii.
[2] *A. f. P.*, xxx. and xxxviii. [3] *M. f. P.*, iii. [4] *N. C.*, 1903. [5] *Z. f. P.*, lviii.
[6] *N. C.*, 1903. See also Hey, "Das Gansersche Symptom," Berlin, 1904.
[7] *Z. f. P.*, Bd. lxi.
[8] See Donath, *A. f. P.*, Bd. xxxii. and xlii. Earlier literature in Denonné, *Thèse de Lyon*, 1894.
[9] *Z. f P.*, Bd. lx.
[10] *Jahrb. f. P.*, xxiii.; see also Leopoldt, *Z. f. P.*, Bd. lxii.; Woltär, *Jahrb. f. P.*, Bd. xxxvii.

mining this point have been described by Fürstner and Wollenberg. The degree of mental confusion and of amnesia following the attack should practically determine the diagnosis.

Tremor is a symptom of hysteria which deserves special consideration, as so many forms may occur which may be confused with the tremor of other diseases.

It is often of a *vibrating*, rapid nature. This is not peculiar to hysteria, but is the effect of the *general nervousness* which so often accompanies it. Another form is characterised by oscillations of average rate and large amplitude, of which there may be 5 to 7 per second. Although this tremor may persist during rest and be specially aggravated by mental excitement, there are some cases in which it is brought on or greatly increased in intensity by active movement. A resemblance to the tremor of sclerosis is thus produced. Here, however, the tremor is not so closely associated with voluntary movements as in sclerosis ; it lasts longer than the movement, occasionally comes on during rest, and may fail to appear during some movement. A form corresponding to the tremor of paralysis agitans has also been described, but in cases which were complicated and not positively hysterical. The tremor is often of an indefinite, inconstant character (polymorph). It may be constantly present, or come on in attacks. Sometimes it is very slight, at other times so severe that it develops into a *clonic spasm*. If the legs are chiefly affected, the tremor may hinder walking. On the other hand, I have occasionally found this tremor and shaking of the legs to be most severe when the patient was lying on his back. It could always be diminished by mental influence, especially by hypnotising the patient. The tremor may be limited to one or more extremities, *e.g.* the arm and leg of one side, or it may extend to the whole body.

The tremor may, even in childhood, reveal the neuropathic constitution.

As regards the hysterical form of myoclonia and general tic, see the corresponding sections.

Hysterial contracture, i.e. fixation of the extremities in certain positions by persistent muscular tension, develops either spontaneously or after attacks of spasm, dreams, pain, or paralysis. It is often limited to one extremity or to a part of it, *e.g.* to the hand or foot ; it sometimes involves the arm and leg of one side or both legs, less frequently all four limbs. It may even occur at the various sites, and the outstanding feature of the condition may be the contracture—the " diathèse de contracture "—evoked by every stimulus (Charcot, Pitres, Mirallié).

The *nature of the contracture*, the *deformities* thus produced, and the *reaction* to *mental* influences are characteristic. It should specially be noted that the muscular tension is immediately increased by the attempt, usually painful, to overcome it, indeed often by a mere touch on the extremity, and that in contrast to the contracture which accompanies true hemiplegia the attempt to approach the points of origin and insertion of the muscles to each other does not aid them to relax. If the arm is affected, it is usually adducted at the shoulder-joint and flexed at a right or acute angle in the elbow-joint, whilst the hand is strongly flexed, in rare cases over-extended, and the fingers are either clenched or fixed in the writing position (as in tetany and paralysis agitans (Figs. 404 and 406). I have, however, observed an extension contracture of the hand and fingers.

There is usually an extension contracture in the legs. The leg is firmly extended at all the joints; even the ankle shows a marked plantar flexion, and the toes are usually flexed, much less often over-extended. In hysterical paraplegia and paraparesis, I have sometimes seen flexion contracture (Fig. 405), or flexion contracture in one, and extension contracture in the other leg. There may also be an equino-varus position of

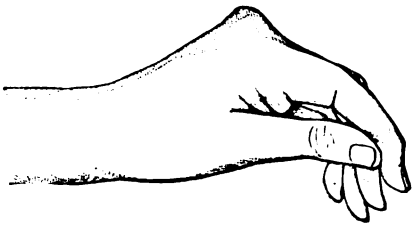


FIG. 404.—Hysterical contracture of the left hand. Drawing by P. Richer. (After Charcot.)

the foot, with marked contraction of the tibialis anticus, but usually with flexion contracture of the toes. The contracture may be limited to a definite group of muscles, *e.g.* to the interossei, the muscles of the neck (hysterical torticollis), etc.

Jolly mentions an interesting case in which contracture of the muscles of the throat and neck was so severe and persistent that a compression paralysis of the ulnar nerve developed with R.D.

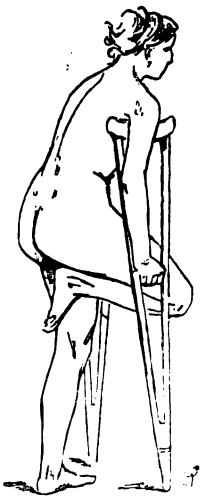


FIG. 405.—Hysterical flexion-contracture of right leg. (After Richer, from the work of de la Tourette.)

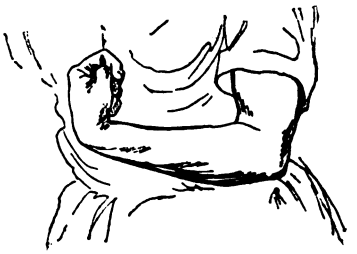


FIG. 406.—Hysterical contracture of the left arm. (After Richer, from the work of de la Tourette.)

Hysterical trismus, i.e. contracture of the jaw muscles, following an injury, has been mistaken for tetanus (Eiselsberg). In one case the condition persisted for nine months (Bidlot-Francotte [1]).

Hysterical scoliosis is usually due to contracture. It affects the muscles (erector trunci, quadratus lumborum) of the side towards which

[1] *Journ. de Neurol.*, 1897.

the concavity of the spinal column is directed. Its characteristics are absence of torsion of the vertebral bodies and of the tubercles of the ribs, and the fact that the condition usually appears and disappears suddenly, and that a change in the position of the body is often sufficient to make the scoliosis disappear. In most cases the whole spine is involved in the curvature, but the scoliosis may be limited to one segment of it. Salomonson points out that it is a secondary symptom and may be the result of an abnormal attitude of the hip—Richet's " station hanchée," *i.e.* the position which comes from resting upon one leg, a position which may produce a slight physiological scoliosis. In many cases hysterical scoliosis is merely an exaggeration of this condition, and is not caused by any primary muscular contraction.

In one of our cases (see Figs. 407, 408), I thought that a habit fixation (in Ehret's sense) was present in addition to the mental factor. The scoliosis had developed in consequence of a

FIG. 407. — Combination of hysterical and habit-scoliosis. (Oppenheim.)

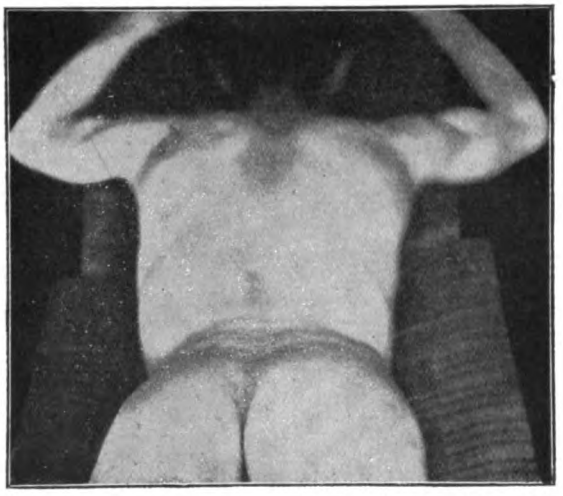


FIG. 408.—The same case as in 407, in the abdominal position. Disappearance of scoliosis.

painful condition which resulted from the lifting of a weight and affected chiefly the left lumbar region. As shown by Fig. 407, the spinal column was so bent that the convexity of the dorsal portion was pushed to the left, the trunk displaced much to the left against the pelvis, the upper part of the body bent to the right and forwards, and there was a deep incurvation above the right ilium, etc. The left half of the thorax stood almost entirely still in respiration, whilst the right side was expanded in the normal way. The skin of the left lumbar region was hypersensitive to pain, and Brodie's symptom was present. The deformity almost entirely disappeared when the patient assumed the abdominal position (Fig. 408).

The mode of production was probably as follows : lifting the weight caused traction upon the lumbar roots of the left side, and possibly hæmorrhage into their sheaths. The pain gave rise to the effort to expand the intervertebral foramina of the left lumbar region, and to keep the left half of the thorax fixed. This attitude had gradually become established by use and wont and by mental influences.

Among the writers who have studied this subject we may mention Duchenne, Brodie, Albert, Guillemet, Vic (*Thèse de Paris*, 1892), Richet-Souques, Germant (*Inaug. Diss.*, Berlin, 1897), Hoffa (" Lehrb. d. orth. Chir.," 1898, and " Die Orthop. im Dienste d. Nerv.," 1900), Wertheim-Salomonson (*Z. f. N.*, xix.), Paosli (*W. m. Pr.*, 1901), Riedinger, Muskat (*C. f. Gr.*, 1901),

Déléarde (*Gaz. des hôp.*, 1902), Heinrich (*D. m. W.*, 1906), Seiffer (*Charité-Annalen*, xxviii.), Zésas (*Arch. intern. de Chir.*, i.), and Strauss (*Mitt. aus d. Grenzgeb.*, xvii.).

The *contractures of the joints*, which are usually associated with *arthralgia* (neurosis of the joints) or hyperæsthesia of the joints, or which by their painfulness simulate an articular affection, are of special interest. The *knee-joint* is most often involved, next the *hip*, and not infrequently the shoulder.

The affection usually commences with pain, which, if the hip-joint is involved, is felt there and at the knee. As a rule it spreads beyond the joint and radiates towards the sacral, lumbar, and inguinal regions. There is no tendency to nocturnal exacerbations. If the muscles surrounding the joint become contracted, the extremity may assume the position and attitude of true coxitis, and the resemblance with this condition may be so great that operations have been performed even by experienced surgeons. The diagnosis is not, as a rule, difficult. Both the tenderness to pressure and spontaneous pain affect the joint and bones less than the *soft parts*; *lifting up a fold of skin* over the joint may even be more painful than pushing the head of the joint into the socket (Brodie). Moreover, this tenderness to pressure extends far beyond the vicinity of the joint. The contracture generally implicates more or less *all the muscles* of the extremity. Finally, the pain and tenderness, and sometimes the contracture, diminish when the patient's attention is distracted. The patient is either quite unable to walk, or, if he can do so, his gait is peculiar and shows an exaggerated limp. The skin round the joint, though usually unaffected, may be red and somewhat swollen, but never in such a way as to resemble an abscess. The temperature is normal.

In doubtful cases *chloroform narcosis* (or, when possible, *hypnotism*) should be used to make the diagnosis. The muscles relax, and it is soon apparent that the joint is free. Slight roughness and perhaps adhesions may develop later in the cartilage. In rare cases where the hysterical contracture has lasted for a very long time, the muscles may become slightly shrunken (myogenic contracture), which causes a shortening that does not disappear during narcosis. The diagnosis depends chiefly upon the presence of other symptoms of hysteria, in particular the disturbances of sensibility in the same side, which are hardly ever absent.

Hysterical contracture may disappear spontaneously, after mental excitement, after spasms, or from the effect of mental treatment. I have observed hysterical contracture of the legs, which had lasted for over a year, suddenly disappear when the patient attempted, in a state of hallucinatory delirium, to jump out of a window.

This contracture is of such a nature that it can be voluntarily imitated. To do this requires, however, great attention, strength, and energy, and Charcot has shown that the strain of the effort produces an irregularity of movement and respiration which is absent in corresponding hysterical conditions. Hysterical contracture may persist during sleep, but this is unusual. On the other hand the contracture of organic brain diseases, *e.g.* of hemiplegia, may disappear during sleep. In many cases there is merely a tendency to contracture, which may be cured by rubbing the skin, pressure, massage, etc., and by vigorous active movements (Charcot, Richer).

Paralysis.—Muscular weakness or pseudoparesis, general or limited to the various limbs, is very common. Although the patient is visibly

exerting himself the voluntary effort is insufficient. This is possibly due sometimes to *want of energy*, or weakness of will power. He does not give one the impression of making any effort; he holds the physician's hand lightly in his own, instead of grasping it firmly, and if by trying to withdraw one's hand forcibly from his, one brings out a response, to a certain extent reflex, one can observe a distinct increase in the strength which he exerts. At other times the feebleness of his movements is due to the fact that the voluntary impulses are not properly distributed, but are diverted to an excessive extent either to the antagonists or to some other muscles, *e.g.* to those of the shoulder in pressure of the hand. Hysterical paralysis is never limited to a single muscle or to the muscles supplied by one nerve. It always affects whole *limbs*, parts of the body which, as the patient imagines, form a whole, or a unite, or involves associated movements which carry out some special function (speech, voice, etc.). It is *never* associated with degenerative atrophy. The muscles generally retain their normal size, or after a long interval show some amount of the atrophy of inactivity. In rare cases there is early development of more marked atrophy (*atrophie en masse*), (Babinski,[1] Massalongo), but in these there is never any qualitative disturbance of the electrical excitability. Moreover, I have frequently seen a true, marked atrophy in the traumatic and vasomotor neuroses, but never in pure uncomplicated hysteria.

Paralysis of the limbs takes the form of *monoplegia*, *hemiplegia*, or *paraplegia*. It develops suddenly after emotional excitement, pain, convulsions, or injuries. If it is complete, the extremity, when passively raised, falls lifelessly back and is entirely out of the control of the patient's will. But it can almost always be ascertained that only *conscious voluntary* movements are impossible, whilst the automatic, emotional reflex movements are more or less retained. The extremity can therefore be used in gesticulation and moved when the patient is under the influence of alcohol and chloroform. I have also found that if we grasp and manipulate the paralysed hand, a slight contraction may sometimes be felt. The patient will specially tend to use his muscles if he has to support himself, *e.g.* if he is in danger of falling. Terror and anger may also contract the paralysed muscles. The arm, if passively raised or brought into a certain position, will often for a moment remain in it—an action which is only made possible by participation of the paralysed muscles.

In one case the patient could not raise her leg from the bed; I passively flexed the thigh, supporting it with the hand, and asked her to make movements of resistance in the ankle-joint. Then I suddenly withdrew the hand which supported the thigh, and the leg remained in this position, owing to the fact that the patient had concentrated her whole attention on the movements of the foot.

If we oppose great resistance to the movement of a group of paresed muscles, *e.g.* the extensors of the knee, and suddenly withdraw it, the limb does not immediately become extended, as in true paresis, but remains flexed, the patient having spasmodically contracted the antagonists (Hösslin [2]). This symptom is frequent but not constant in hysterical

[1] *Prog. méd.*, 1886. From the writer's discussion, I am not clear how he interprets the results which he reports. In any case he will not admit that trophic disturbances can be a symptom of hysteria.

[2] *M. m. W.*, 1902.

paralysis. Hösslin is therefore wrong in regarding it as a criterion of simulation.

A pure *monoplegia, i.e.* incomplete total paralysis of one extremity, with normal movement of the other limb on the same side, occurs only in hysteria. The paralysis may be limited to one hand or foot.

Hysterical hemiplegia seldom involves the facial and hypoglossal muscles. There is sometimes marked or exaggerated deviation of the mouth and tongue, caused by contraction of the muscles of the opposite side (hemispasmus glosso-labialis, Fig. 409). The contracture may also involve the muscles of the hemiplegic side, the deviation being then towards the opposite side from that in true hemiplegia. The presence of a spasm of this kind is shown by the tension of the muscles, and in the tongue by the fact that, if by using great force we succeed in pushing it towards the opposite side, it immediately resumes its extreme position. It may also be so strongly curved inside the mouth that it cannot be protruded. The half of the tongue involved in the spasm seems thicker and smaller. The cheek is not blown out in expiration. The contracture may also be recognised from the folds round the angle of the mouth and the fibrillary tremor. In true facial paralysis a light held before the mouth is most easily blown out on the affected side.

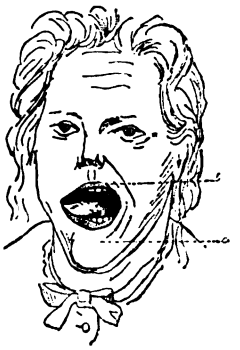


FIG. 409. — Glosso-labial hemispasm in hysteria. Fibrillary tremor at *b* and *c*. (After Charcot.)

True paresis of the corresponding facial is extremely rare. It is often impossible to determine how much is paralysis, how much contracture. I have once observed a *glosso-labio-maxillary hemispasm.* Sachs,[1] who has lately studied this subject thoroughly, points out the ideogenic origin of all these disturbances. Ziehen states that hysterical hemispasm is often associated with ptosis or pseudoptosis on the other side, causing a kind of alternate hemiplegia. I have not so far seen this condition.

Hysterical monoplegia and hemiplegia is either flaccid or combined with contracture which shows the characteristics just described. Although the tendon reflexes are occasionally exaggerated, the characteristic symptoms of muscular rigidity (see pp. 180 and 808), and notably the Babinski and Oppenheim signs, are absent.

A good description of hysterical hemiplegia and the symptoms which differentiate it from the organic form is given by Babinski (*Gaz. méd. des hôp.*, 1900).

As Todd long ago recognised, the *gait* does not correspond to that in true hemiplegia. The leg is simply dragged, not moved in a circle, or the patient leans on a crutch and does not touch the floor with the foot. We often find the foot dragged along with the whole of the sole or heel on the floor. The gait is sometimes like that of a child walking on stilts.

Hysterical paraplegia is usually associated with contracture.

Abasia, i.e. absolute inability to walk and usually also to stand, the movements being normal in the recumbent position, is not uncommon. There are also many forms of hysterical disturbance of gait (dysbasia hysterica), which may imitate almost every type due to organic disease, but

[1] *A. f. P.*, Bd. xlii.

are usually distinguished from them by certain features. The hysterical origin is chiefly indicated by the exaggeration and forced character of the movements, by the fact that these movements may be normal under other circumstances, *e.g.* in the recumbent position, by the disappearance of the disturbance under mental influence, or when the attention is diverted. One of my patients could run, but could not walk; another could run better backwards than forwards, etc. A common form is a kind of spastic gait, but without the muscular stiffness. The patient does not usually walk upon the toes, but upon the whole sole or the heel. The gait in many cases resembles that of cerebellar ataxia, but the movements are tortuous, like those of a snake, or the tottering has a forced character, which shows its hysterical nature. In one of my cases which followed influenza, the waddling gait was exactly like that of dystrophy; the patient could not keep the trunk erect in sitting, but had to support it with her hands. The paralysis was localised in the lumbar-pelvic-thigh muscles, but there was no atrophy or pseudo-hypertrophy, and the influence of suggestion made its hysterical nature evident. The disturbance of gait is more often caused by painful contracture of the lumbar-pelvic muscles.

The *tendon reflexes* are normal or even exaggerated. This exaggeration is often not real; the mechanical muscular contraction is followed by tremors in the quadriceps and other distant muscles, which are an effect of mental excitement and disappear when this subsides or the patient's attention is diverted. The foot clonus in hysteria is usually also not genuine. The various contractions follow at irregular intervals, the tremor is rapidly exhausted, and is less dependent on mechanical tension of the muscles than on the mental factor, etc. A foot clonus which cannot be definitely distinguished from true clonus may, however, occur in hysteria (see further on). The knee-jerks are practically never absent, although two recent cases reported by Nonne [1] show that under unusual conditions, which cannot now be ascertained, they may disappear. Nonne refers to a couple of similar cases described by other writers.

Hysterical aphonia is the most common form of hysterical paralysis, the voice being most intimately connected with the emotional life. It almost always comes on *suddenly*, as the result of the causes already mentioned, more especially some mental excitement. It may also be due to some reflex cause, such as catarrh of the larynx. The voice is generally completely lost, the patient speaking only in whispers. Laryngoscopic examination shows that on the attempt to phonate, the vocal cords either cannot be brought out of the position of abduction or can only be partially adducted. The internal thyroarytenoid is sometimes paralysed. Less often the glottis shows the position characteristic of paralysis of the transverse arytenoid muscle. Unilateral paralysis of the vocal cords hardly ever occurs. The laryngoscopic pictures are such as might be voluntarily produced. Coughing and hawking are usually accompanied by vocal sounds, and expressions of pain may also be. Some patients are even able to sing. Anæsthesia or hypæsthesia of the pharynx and entrance to the larynx are often present. *Hoarseness*, usually associated with a piping voice, is less common than hysterical aphonia.

The aphonia tends to disappear suddenly and *spontaneously*. (This is usually, however, the effect of emotion. One of my patients who had suffered for a long time from aphonia, one evening when the moon was

[1] *Z. f. N.*, xxiv. See further remarks on this subject in section on differential diagnosis.

shining into her room, repeated Goethe's poem : " Füllest wieder Busch und Tal," etc. She was thrilled with emotion, and by the third line she had found her voice.) It may also be cured by mental excitement or by treatment. It is apt to *relapse* easily and to become more intractable with time.

Mutism, or hysterical dumbness, is less common. This condition, which has been specially studied by Charcot and Cartaz, is characterised by complete inability to form sounds. The patient makes a great effort, but he cannot produce a tone, nor even a whisper. When he attempts to speak the lips and tongue seem to be completely paralysed, although they may be moved otherwise, or they may be distinctly contracted without producing any sound. It is a characteristic feature that the patient understands everything, and is yet more dumb than an aphasic, who can generally utter a few sounds or words. Moreover, his power of facial expression (in contrast to that of the aphasic) is very marked. Phonation may be unimpaired and the mutism incomplete. The patient, with a visible effort, pronounces the first sound of a word, then after a pause the second, but no more. He thus differs from the aphasic who can either say nothing, or continually repeats the same sounds and words. This form marks the transition to *hysterical stuttering*, which usually develops from (or precedes) mutism, and leads to normal speech. An individual with mutism can read and write. I have seen one exception to this rule in a mute hysteric who could not write. Charcot, Ballet, and Sollier have described cases of this kind. The attack is generally brought on by fright. In two of my cases the loss of speech was brought on by lightning which struck the ground close to the patients, in another by the sight of a mouse. It may pass away in some hours, days, weeks, etc., but may persist for years. As I have already mentioned, hysterical mutism may be associated with deafness.

Raymond[1] has described a form of word-blindness due to hysteria, and refers to cases by Wernicke and others.

In a few cases (Raymond, Janet, Schnitzer, etc.) hysterical speech disturbances are said to have simulated motor aphasia, paraphasia, etc. But my opinion in opposition to that of Mann (*B. k. W.*, 1901) and Guillain is that we cannot be too careful in our interpretation of such cases.

Hysterical *stuttering*, which may be independent of mutism, may resemble the ordinary form, or each word may be broken up into very irregular portions ; the patient repeats the same sound or syllable several times, the others following after a longer or shorter interval, a word being shot out explosively every now and then. The action of the muscles which accompanies it seems to be somewhat forced. Respiratory symptoms may be absent, or, like the contractions of the facial muscles, they may persist when the patient is at rest. The absence of intermissions may also be regarded as characteristic of the hysterical nature of this speech disturbance.

A speech disturbance akin to the syllable stumbling of paralytics has been observed in rare cases of hysteria.

On this subject see Bödeker, *Charité-Annalen*, xv.

Hysterical *paralysis of the soft palate and muscles of deglutition* is

[1] *R. n.*, 1899.

extremely rare, and should only be diagnosed when there are convincing reasons for believing that the patient is hysterical and when every complication can be excluded. It was formerly often mistaken for myasthenic paralysis (*q.v.*). Paralysis of the soft palate causes nasal speech. The dysphagia is due, as already stated, either to a spastic contraction of the œsophagus or cardia, or it represents a mental symptom, *i.e.* a phagophobia. I have treated a patient who was fed for months with a tube. If fluids were forcibly placed in her mouth, they flowed out again. Rossolimo has published interesting cases of this and a similar kind, but the affection which he calls dysphagia amyotactica is not always hysterical in its nature. In hysterical dysphagia the articulation may be quite normal. The palatal and pharyngeal reflexes are often absent, but according to Stursberg [1] this is almost as often the case in healthy persons.

The *muscles of the eye-ball* are seldom paralysed. There may be ptosis, which almost always proves to be a *pseudo-ptosis* due to spasm of the orbicularis palpebrarum. On attempting to raise the eyelid one feels the muscular contraction and sees a contraction or fibrillary tremor of the muscles of the eyelid. This pseudo-ptosis does not diminish when the patient throws back his head. In distinction to true ptosis, the eyebrow is generally lower than that of the normal side. Paralytic ptosis is a much less common condition. It cannot always be absolutely distinguished from true ptosis, as Hitzig, Saenger, and I have found. The secondary contraction of the frontalis, so characteristic of true ptosis, is certainly absent (see Figs. 399, 400).

See also the paper by Sauvineau, *R. n.*, 1907.

In a few cases there is a spasm of the levator palpebræ superioris, which gives a very characteristic expression (of astonishment) and which may produce Graefe's and Stellwag's signs. I have seen cases of this kind in which it was not always possible to give a definite diagnosis of hysteria. Nor can we always determine whether the spasm involves the levator or Müller's muscle.

The most frequent hysterical disturbance of vision is *diplopia*, or *monocular polyopia*. When one eye is closed, there is double or multiple vision in the other. This can be demonstrated by asking the patient to fix an object which is gradually moved away from his eye. A double image appears at a distance of 10 to 15 cm., and at a greater distance is joined by another, close to or above it. This is probably due to defective accommodation, in particular to a spasm of the muscle of accommodation (see p. 78). It may be combined with *micropsia* and *macropsia*, symptoms which may also be due to other causes, such as epileptic aura, etc. Parinaud speaks of micromegalopsia. There is undoubtedly a hysterical form of spasm and paralysis of the muscle of accommodation. In one of our cases the paralysis was of an intermittent character.

Among the recent works on micropsia, see those of Pfister (*N. C.*, 1904) and Heilbronner *Z. f. N.*, xxvii.).

Diplopia is usually due to *contracture* of the extrinsic muscles of the eye. We have little reliable knowledge as to hysterical *paralysis of the ocular muscles*. It is generally simulated by contracture of the antagonists. Spasm of the internal rectus, for instance, may simulate paralysis of the externus. Paralysis of conjugate deviation may be due to contracture. Forcible contraction of the internal recti may even resemble

[1] *M. m. W.*, 1902.

external ophthalmoplegia. On the other hand, the associated lateral movements may sometimes be carried out, not voluntarily, but automatically, as the result of habit, etc. There is, of course, a cerebral form of ophthalmoplegia (see p. 699), of which this is also true to some extent.

From my own experience, I would unhesitatingly deny the existence of true hysterical paralysis of the ocular muscles, especially the paralysis of one individual muscle and of the internal muscles of the eye. There are, however, cases, no longer isolated examples, which obviously prove that an apparent paralysis of the ocular muscles may accompany hysteria. Amongst these I would cite the cases of Wilbrand-Saenger, Roeder, Donath,[1] Nonne-Beselin,[2] Hitzig,[3] Liebrecht,[4] A. Westphal,[5] Karplus,[6] B. Sachs, Weiss, Cramer, and Pichler. This seems also to be recognised with regard to myosis,[7] mydriasis, paralysis of the muscle of accomodation, and rigidity of the pupils (in the wider sense of the word). A spasm of the antagonists may also simulate a condition of paralysis. Spasm of the sphincter pupillæ, for instance, may appear to be immobility of the pupils. A communication by A. Westphal is very interesting in this respect. He was able to show that immobility of the pupils combined with myosis was only present in hysteria when the patient's attention was concentrated on the test, the pupils dilating and reacting when the examination was made without her knowledge. In many other cases this symptom was influenced by suggestion, transfer, etc. A. Westphal has observed loss of the pupil reflex during hysterical attacks in a patient who afterwards became epileptic—a case of special interest. Karplus, Bumke,[8] and Schultze[9] have all observed immobility of the pupils during hysterical attacks; this symptom can therefore no longer be regarded as occurring exclusively in epilepsy. Steffen has also called attention to this fact.

Even now, however, I think it advisable to regard the occurrence of true paralysis of the ocular muscles in hysteria as very doubtful.[10] *I believe especially that persistent reflex immobility of the pupils is not a symptom of hysteria*, although it may be *associated* with it. Saenger as well as Hoche, Parinaud, Marburg, and later Bumke and Ziehen[11] have expressed this view. Saenger found that in some cases the immobility of the pupils, due to spasm of the sphincter, disappeared when the patient was kept in a dark room. One should always remember the possibility of a complication and of simulation (from the use of atropin).

[1] *Z. f. N.*, ii.

[2] "Kontraktur und Lähmungszustände der ext. und int. Augenmuskeln bei Hysterie." Festschrift, Hamburg, 1896.

[3] *B. k. W.*, 1897.

[4] *A. f. Aug.*, Bd. xxxiv.

[5] *B. k. W.*, 1897, and *D. m. W.*, 1905; *C. f. N.*, 1906.

[6] *W. k. W.*, 1896.

[7] I have seen marked myosis with normal reaction of the pupils occur in a few cases in which the other climacteric symptoms pointed to a sympathetic origin.

[8] *M. m. W.*, 1906.

[9] *Therap. d. Geg.*, 1907.

[10] I have seen two almost similar cases of grave hysteria in which nystagmus and trochlear paralysis came on suddenly with vertigo and sickness. Although all the other symptoms were open to mental influence, these proved incurable. No doubt they were caused by some focal disease, probably a hæmorrhage, which had occurred at the height of an attack of hysterical vomiting; in the other case they were possibly due to a delicacy of the vasomotor system. The symptoms were certainly produced by the hysteria, but were not proper to it. If we remember the richness of the vascular plexus in the aqueduct of Sylvius and the narrowness of the passage at this point, we can well understand that great fluctuation, accompanied by corresponding dilatation of the vessels, would be sufficient to produce grave functional disturbances of this region.

[11] *Die Deutsche Klinik*, etc., 1906. Article on Hysteria.

We should refer to Bumke's careful study of the matter, "Die Pupillenstörungen bei Geistes- und Nervenkrankheiten," Jena, 1904, and to his article in *M. m. W.*, 1906. There is a very interesting account by Redlich (*D. m. W.*, 1908), of a patient who suffered from immobility of the pupils during hysterical attacks, and who in the intervals could not only effect physiological dilatation of the pupils, by strong and persistent action of the muscles, but could also inhibit the reaction and thus prevent the pupils contracting for light.

Insufficiency of the internal rectus (Landolt) is a common symptom. True *nystagmus* is not a symptom of hysteria. A kind of rapid oscillatory-nystagmus may occur (Sabrazès[1]), as I have seen, which may be influenced by reflex (light stimulus) and mental factors, and may be associated with blepharospasm. I have occasionally found that when one eye is closed, the other twitches convulsively and assumes the extreme position of convergence, the pupil being greatly contracted.

Hysterical *paralysis of the diaphragm* is very rare. It may be easily diagnosed from its sudden onset following upon some mental excitement, the marked difficulty of respiration, even when the patient is in repose, the excessive retraction of the abdomen during inspiration, and other well-known hysterical factors. It should be remembered that symptoms of paralysis of the diaphragm, especially inspiratory retraction, may be voluntarily simulated. Wernicke regards insufficiency of the diaphragm as a common occurrence in hysteria and neurasthenia. It plays an essential part in nervous asthma. Barth describes phrenic asthma due to hysteria.

Incontinence of urine, caused by anæsthesia of the mucous membrane of the bladder and urethra, is exceedingly rare. *Retention of urine* and *dysuria* are more common; they are caused by spasm of the sphincter. Catheterisation is thus rendered difficult and *painful*. Strangury, with frequent passing of small quantities of urine (polyuria) sometimes occurs. Urinary troubles which belong to the category of fixed ideas and phobias, may accompany hysteria, but will be discussed at another place.

One of my patients was obliged to pass urine every time he thought of the process; if he did not yield to the pressure, the urine passed involuntarily. When his attention was otherwise engrossed, the urine was normally retained. Incontinence in conditions of anxiety may be a symptom of the neuropathic diathesis. See also Frankl-Hochwart and Zuckerkandl; also Hock (*D. m. W.*, 1906).

Gastric Disorders.—Loss of appetite frequently occurs, sometimes alternating with bulimia. This may become so marked that at first some kinds of food, and later all food is refused, no nourishment of any kind being taken (*anorexia hysterica*). This condition is not without danger if not treated in time. Pain is seldom given as the reason for refusing food. Mental processes are apparently the chief factors in the production of anorexia.

I have seen a lady in whom an inanition psychosis followed marked hysterical anorexia. She recovered after being treated in a sanatorium at my recommendation, gaining 45 lbs. in weight from her forced feeding.

Hysterical *vomiting* is more frequent. The food is vomited in an undigested condition, almost immediately after it is taken. Mucus and saliva are also vomited. The appetite, however, usually remains good and the patient does not become emaciated. Urine has occasionally

[1] *Semaine méd.*, 1894. Also Stransky, *N. C.*, 1901.

been found in the vomit. This disorder may be very persistent, but is seldom dangerous. In one case observed by Ewald, the vomiting ceased after a gastroenterostomy was performed. The vomiting may be due to spasm of the pyloric or cardiac orifice. The tendency to vomiting was so marked in a lady under my care, that eructation and vomiting occurred every time she brushed her teeth, touched her throat, or washed her face. Morning vomiting may be a symptom of hysteria.

The varieties of hysterical vomiting have been described by Mathieu-Roux (*Gaz. des hôp.*, 1906). Rumination (merycism) may occur, but it is a symptom of the neuro-psychopathic diathesis.

Obstinate constipation is common. Diarrhœa sometimes follows mental excitement. The intimate relations between membranous enteritis or mucous colitis and hysteria (as well as neurasthenia) have long been recognised.

Several of my patients have complained of weakness after evacuation of the bowels.

In many cases marked protrusion of the abdomen, caused by accumulation of gas in the intestine (tympanitis, *meteorism*), is one of the chief complaints. Contracture of the diaphragm and flaccidity of the abdominal muscles has also been regarded as the cause (Talma, etc.). Headache, vomiting, and other disorders may be combined with it. In a case of Huchard's, meteorism was said to be the cause of death, but this is an extremely rare occurrence. On the whole it is a harmless symptom and passes off without any evacuation of gas. Hysterical meteorism tends to disappear when the patient is under an anæsthetic (Spencer Wells, Talma). A loudly audible noise in the intestine (rumbling) is also an hysterical symptom. Hysterical meteorism may, to superficial examination, simulate an abdominal tumour or pregnancy.

Compare Henry, "Les fausses grossesses," *Thèse de Paris*, 1904; and Beck, "Abdominale-Scheingeschwülste bei hyst. Kind.," *M. f. Kind*, 1905.

Fœcal vomiting, with symptoms of "pseudoperitonitis" or intestinal obstruction, has been frequently observed (Cousot, Bristowe, Sanders, Schloffer, Bregman, Weber,[1] Kausch,[2] etc.). Nothnagel has tried to explain it as being due to antiperistalsis (drawing his conclusions from the cases of Briquet and others). It has already been shown that the cause is an intestinal spasm.

Anomalies of Secretion.—Salivation (an interesting case is described by Brissaud and Brécy), profuse perspiration, coryza, and rhinorrhœa, dacryorrhœa, etc., and unilateral hyperidrosis may be present, but these are not symptoms peculiar to hysteria. Curschmann[3] has lately described profuse sweating in hysterics. Sweat, tears, and saliva of hæmorrhagic nature have also been observed. Xerostomy may be a symptom of hysteria. Pawlow has emphasised this and pointed out that the glands may react to psychical as well as to physiological stimuli. Transient polyuria may follow the attacks, or diabetes insipidus may be associated with hysteria.

In some cases there is marked *oliguria*, which may increase to anuria. The fact that uræmia does not develop, even when this symptom is of long duration, has been ascribed to a vicarious activity of the stomach,

[1] *Br.*, 1904. [2] *Mitt. aus d. Grenzgeb.*, xvii. [3] *M. m. W.*, 1907.

urine being found in the vomited matter (Charcot, Guisy, etc.). The physician is, however, often led astray by the patient.

See also Frankl-Hochwart; also Cestan-Noguès (*R. n.*, 1905). Nephrectomy has been performed in several of these cases (*e.g.* by Pousson, *R. n.*, 1907), and the kidneys found normal.

I have seen a case in which nephropexia was performed on account of severe pain which was attributed to floating kidney. This was followed by attacks of acute pain with oliguria, and it was found by catheterisation of the urethra that the corresponding kidney secreted only some sixteenth part as much as the normal one. It was thought that the kidney might be compressed, but this was not found on operation to be so, and the patient, who suffered from grave hysteria, gradually recovered.

In a few cases galactorrhœa has been observed.

Vasomotor and Circulatory Disturbances.—Rapid change in the colour, sudden pallor and flushing, are common in hysteria, but by no means peculiar to it (see chapter on neurasthenia). *Cyanosis* occurs in the paralysed and contracted limbs. It may be associated with swelling, which is mistaken for œdema, although pressure of the finger does not leave pitting. This swelling is usually hard, elastic, and combined with decrease in the temperature of the skin. This condition has been termed "blue œdema." Another form of œdema, in which the skin is pale and bloodless ("œdème blanc"), may be present. The temperature of the skin is rarely increased in hysterical œdema, as in a case described by Raymond-Cestan, which was remarkable for the transformation of the blue œdema into white. True œdema, usually of an acute circumscribed character (*q.v.*), is seldom found. A swelling of this kind is sometimes localised over the joints (*e.g.* of the fingers), is associated with pain on movement, and simulates a joint disease. The forearm is often affected. Involvement of several limbs is less common.

A disturbance allied to intermittent claudication (see p. 586) may also develop from hysteria, as shown by a case of Westphal's (*C. f. N.*, 1905).

Local *asphyxia*, *factitious urticaria*, and many other cutaneous affections (erythema, pemphigus, and even gangrene) may develop. These may, however, often (some writers think always) be due to voluntary injuries. So-called "multiple neurotic gangrene" (Doutrelepont, Kaposi, Cassirer) is mostly observed in hysterics and is often attributed to voluntary injury (*e.g.* by Bettmann[1]). An atypical zoster (Bettmann, Kreibich) and a form of purpura (Lancéreaux) have also been regarded as hysterical.

On the subject of neurotic gangrene, see Le Gall (*Thèse de Paris*, 1902), Janvier (*Thèse de Paris*, 1902), and Zeiler (*Z. f. N.*, xxviii.), who found a distinction between artificial and spontaneous necrosis in the microscopical condition of the diseased skin, the cutaneous surface being mainly injured in the former, and the cutis primarily affected in the latter. See also Kreibich, *Dermatol. Zeitschr.*, 1904, and Róna (*Orvos. Hetil.*, 1905). The discussion on hysteria in the Neurological Society of Paris (*R. n.*, 1908) should also be consulted.

It is often noticed that patients with hysterical anæsthesia do not bleed from deep prick of a needle. In a small operation (excision of muscle) which I performed on the anæsthetic arm of an hysteric, there was no bleeding. This symptom, which in hemianæsthesia is not confined to the affected side, is due to a vascular spasm. It is true, however, that needle pricks do not always bleed, even in healthy persons.

[1] *M. m. W.*, 1903.

Spontaneous hæmorrhage on the skin of the forehead, breast, and elsewhere is very rare, and is not always due to deception on the part of the patient. Hæmorrhage from the internal organs is also rare. It most commonly arises from the stomach or uterus. It may take the place of, or accompany menstruation. Lermoyez, for instance, describes bleeding from the ear as vicarious menstruation. Hysterical bleeding from the ear is mentioned by Gradenigo, Gellé, Souques, and others. The hæmorrhage of hysteria may be very profuse, but even then it has a remarkably slight effect upon the general condition (cases of Huchard, Charcot, Senator, Débove, Lancereaux, Holth [1]). Hysterical renal hæmorrhage has also been described (Poljakoff, Klemperer,[2] etc.) Sainton mentions bleeding from the breasts in multiple hysterical hæmorrhage.

Hæmatemesis on a neuropathic basis is discussed by Sée (*Thèse de Paris*, 1901) and Noel (*Thèse de Paris*, 1905).

In a young girl under my care, whose father suffered from neurasthenia with various vasomotor symptoms, there had been, since the onset of menstruation, a constant instead of a periodic bleeding from the genitals, without any local change. Local treatment had no effect, but cold seabathing stopped the hæmorrhage for a time. She suffered also from urticaria, erythrophobia, and a form of hydrops articulorum intermittens.

A young hystero-neurasthenic, affected with unilateral sweating and flushing, found his nose bleed each time he saw a red mass of copper in his factory (he was an electrician).

Vasomotor disturbances may also have the effect of provoking other symptoms. The influence of the mind upon the vasomotor system, which is often greatly increased, explains many symptoms which would otherwise be obscure. I have noticed that this abnormal excitability of the vasomotor system and even of the vomiting centre is comparatively frequent in the children of diabetic parents, but the disorder is more often of a neurasthenic than of an hysterical character.

Acceleration of the pulse, occurring in paroxysms and combined with palpitation of the heart, is a common symptom, but the pulse may be normal in spite of the sensation of palpitation. During the spasm it is normal or accelerated ; in the condition of syncope it is usually retarded.

Bradycardia is occasionally mentioned (Debove, Triboulet, *Trib. méd.*, 1904). See also Bernheim, *Rev. de Méd.*, 1904 ; Galdi, *Il Morgagni*. 1904.

Trophic disturbances are not important. The hair has been observed to fall out or become suddenly grey. Degenerative muscular atrophy does not occur. Simple atrophy (associated with quantitative decrease of excitability) is rarely marked, and I am doubtful if it ever occurs (see above). The trophic disturbances sometimes noticed on the skin have already been referred to. Even perforating ulcer of the stomach has been regarded as a symptom of hysteria (Gilles de la Tourette), a view which we must decline to accept.

The *sexual sphere* is often involved. Impotence and decrease of sexual power, as well as aberrations of the sexual instinct, may be symptoms of hysteria. Loss of desire may be the result of anæsthesia of the vaginal mucous membrane, but is not always due to this cause. This mucous membrane may be hyperæsthetic and may be the site of hysterogenic zones. Many hysterical patients complain of molimina menstrualia, and the other troubles tend to become increased at the time of menstruation.

Metabolism is usually unaffected. According to Gilles de la Tourette and Cathélineau, with whom Rybalkin agrees, it is only altered during the attacks in such a way that the fixed con-

[1] *Norsk. Mag. f. Lägevid.*, 1901. [2] *D. m. W.*, 1897, and *Therap. d. Geg.*, 1901.

stituents of the urine passed in the twenty-four hours following the attack—the urates, phosphates, etc.—are reduced by one-third, and that the proportion between the earthy and alkaline phosphates, which normally is 1 : 3, becomes 1 : 2, and even 1 : 1. These data, however, require to be tested and confirmed, and their diagnostic value has been doubted, even by the writers themselves, who found a similar condition in other diseases.

Only a few cases of *hysterical fever* have been recorded (Toussot, Reviére, Briquet, Chauveau, Sarbo, Dippe, Menzer, Wormser-Bing, Strasser, Vaillard, Larré,[1] Lannois-Porot,[2] Bernheim,[3] Caramano,[4] Voss,[5] Goldflam,[6] Wilmans,[7] etc.). The symptom is, to say the least, exceedingly rare, and care cannot be too urgently enjoined in the diagnosis (bearing in mind the possibility of simulation or complications, *e.g.* tuberculosis, disease of the genitals, etc.).

Recent cases show beyond a doubt that hysterical fever may occur. The fact does not seem so astonishing in view of our knowledge of the intimate relation between the mental life and the vasomotor processes. Dejerine, who recognised hysterical fever, pointed out that young, impressionable individuals might have a rise of temperature after any excitement. I have also observed some rare cases in which simulation was absolutely excluded. Jolly and also Strümpell (*Z. f. N.*, xxx.) have expressed their disbelief in the occurrence of hysterical fever. See also Dircksen, *Thèse de Paris*, 1904, and the discussion in Paris on hysteria (*R. n.*, 1908). I might mention that I have seen a patient who suffered from manic-depressive insanity, and who in some phases of her illness had continued high fever combined with amenorrhœa.

In the cases reported, the fever was either continuous or intermittent. The latter form was characterised by sudden rise of temperature to 42° (107·6° F.) and even to 45° C. (113° F.), by absence of corresponding accessory symptoms (alteration in the pulse, respiration, urine), or want of proportion between these and the rise of temperature, and also by the slight effect of antipyretics. The oscillations from day to day may be very marked, and the temperature may vary greatly at different parts of the body. Other criteria for the differentiation of true from hysterical pyrexia (Stern, Strasser) have proved to be unreliable. Kausch [8] has lately studied this subject with much care, taking all the published cases into his consideration. In spite of a most critical examination he had to admit the existence of hysterical fever.

Increase in the blood pressure has been several times observed in the functional neuroses and specially in hysteria. It may be the effect of exaggerated mental excitability. Cases of this kind, which of late have been examined with Gärtner's tonometer, have been published by Federn, Heim, Kapsamer, Tschlenoff, Hochhaus, Hensen, Strauss, and others.

Grouping of the Symptoms. Development and Course

In examining a large number of hysterical cases, one is astonished at the disparity of the symptoms. Some mild cases show merely slight objective signs; in some a single symptom seems to express the whole disease (*monosymptomatic*), and in others a whole crowd of the above-described symptoms pass before us in a motley variety.

In Germany, the milder cases predominate. The patients are women who complain of great excitability, unrest, globus, anxiety, headache,

[1] *Thèse de Paris*, 1903. [2] *Lyon méd.*, 1903. [3] *Revue méd. de l'Est*, 1904.
[4] *Presse méd.*, 1905. [5] *Z. f. N.*, Bd. xxx. and xxxi.
[6] *N. C.*, 1906. [7] *C. f. N.*, 1908.
[8] *Mitt. aus d. Grenzgeb.*, 3 Suppl., and *Mitt. aus d. Grenzgeb.*, xvii.

etc., and we can base the diagnosis either upon the history of previous convulsive attacks, or upon the objective signs of a sensory disturbance or some other symptom. There may be every transition from these to the most severe forms, which are chiefly characterised by major attacks. In each case some special symptom predominates, and may indeed, for a time, be the only one present. It may be hysterical aphonia, anorexia, tympanitis, or vomiting; in many cases it is pain, in others paralysis or contracture. The symptoms most commonly found are of a spasmodic character. The monosymptomatic forms (contractures, paralysis, abasia, hallucinatory delirium) are most common in childhood. The fact that these symptoms respond most readily to treatment gives an individual stamp to the hysteria of childhood.

If, on examination we find merely local symptoms, and no sign of general disease, we speak of local hysteria; but this is a meaningless term which should be abandoned.

The disease may develop gradually. It commences with subjective sensations, and later, some particularly distressing symptom draws attention to the condition. Or, it may have an acute onset, marked symptoms immediately following some mental emotion.

The course is seldom acute. If we exclude the unusual form of acute fatal hysteria, which indeed should not be considered here, there are a number of cases in which the hysterical symptoms appear, and then vanish in a few weeks never to return. But in the great majority of cases the disease is *chronic*, lasting for many years, even till old age comes on, when it disappears with the climateric or the commencement of senile involution. I have seen it persist in a few cases into the eightieth year of life.

This chronic course is, however, not a steady one. The condition is liable to *great variations* and the symptoms *change* as they do in no other disease. There is sometimes marked alternation in the symptoms, *e.g.* during an attack of hysterical hemicrania, globus may occur, the anxiety and other psychalgias completely disappearing, and so on. The sudden disappearance of the most severe symptoms and the onset of others is particularly characteristic. *Long intermissions*, in which the general condition is practically normal, frequently occur.

Pathological Anatomy.—We are not justified in speaking of a pathological basis for hysteria. In the cases examined after death there were either no change at all, or merely such as represented *congenital anomalies of development*, or the effects of the malnutrition (inanition) due to the hysteria. The course, the instability of the symptoms, their dependence on mental influences, their sudden disappearance and variability are all opposed to the assumption of a pathological basis in the general sense of the word.

The view that *molecular* alterations in the central nervous system, and particularly in the cerebral cortex, are the cause of hysteria would best explain the symptoms. Naturally we cannot produce any convincing proof of the correctness of this view. There must be an exaggeration of those fine differences in the organisation of the central nervous system, which must be assumed to exist even in normal persons in order to explain the differences in the excitability of various persons, sexes, and races. At the same time it is not impossible that these fine changes extend to the whole nervous system. There is absolutely no doubt that disturbances of association play an essential part in this disease, but we are by no means

justified in attributing it forthwith to a lesion or functional inhibition of the association tracts.

It should also be remembered that organic diseases of the nervous system due to congenital errors of development (*e.g.* gliosis) may co-exist with hysteria, and that we may be right in regarding these as the cause of the hysteria. The cases described by Berkley and others are thus explained.

An attempt has been made to ascribe hysteria to anomalies of metabolism, such as abnormal processes of oxidation, etc. (Biernacki, Vigouroux), but this theory has no definite basis.

Differential Diagnosis.—The diagnosis is as a rule an easy matter, but it may be exceedingly difficult. It should always be established *by means of exclusion.* This is absolutely necessary, as the disease is very often associated with other affections, and specially with other organic diseases of the nervous system. After examination of the internal organs, therefore, which should never be neglected, the first step should be to look for symptoms which are only present in organic diseases of the nervous system. These include ophthalmoscopic lesions of the optic nerve, reflex immobility of the pupils, which may be a permanent and isolated ocular symptom, paralysis of a single nerve (oculo-motor, facial, peroneal, etc.), degenerative atrophy, Westphal's sign, and true ataxia.[1]

Nonne (*Z. f. N.*, xxiv.) has lately found the knee-jerk absent in two cases of hysterical paralysis. There can be no doubt as to the cases, but the symptom is in itself so absolutely exceptional (I have never seen anything like it) that I think I am right in advising that it should never be taken into practical consideration. A physician who follows this advice may perhaps once in his life fail to recognise hysteria, but if he regards Westphal's sign as one of the symptoms of hysteria he will be in danger of making a wrong diagnosis at every step. In Nonne's cases the symptom was associated with marked hypotonia, and both these symptoms were of a transient nature. It should also be noted that the patient was born of alcoholic parents. Köster's case (*A. f. kl. M.*, Bd. xc.) does not seem to me fully proved. Wigand (*N. C.*, 1907) describes another case from Nonne's Clinic.

There are other symptoms, which cetainly may occur in hysteria, but are so extremely rare that their presence at least throws doubt upon the diagnosis of hysteria. These are hemianopsia, true nystagmus, bulbar speech disturbance, typical scanning, typical intention tremor, chronic incontinence of urine, internal ophthalmoplegia, etc. Persistent hemianopsia definitely excludes hysteria.

The diagnosis may be specially difficult from the fact that hysteria may not only show some symptoms resembling those of other diseases of the nervous system, but may simulate these diseases in every particular.

The diagnosis from *disseminated sclerosis* (*q.v.*) may be difficult in cases which show no sign of disease of the optic nerve. But since we have learned the peculiar characteristics of the *spastic* condition of the muscles caused by organic disease, and specially since we have known the Babinski and Oppenheim signs, which do not occur in hysteria, the diagnosis from disseminated sclerosis has become more easy and simple. Even in the

[1] A hysterical form of ataxia may occur, but it can always be distinguished from true ataxia. To give an example: one of my patients, when his eyes were closed, always carried the tip of his finger to the eye or forehead instead of the point of his nose, but this was not accompanied by swaying, zigzag movements, or purposeless associated movements, the tip of the finger always going direct to its goal. In hysterical ataxia there is always excessive swaying of the body, and skilful examination will reveal the psychogenic nature of the symptom. In one patient in whom all my efforts had at first proved vain, the tottering disappeared whenever I asked him to look me straight in the eyes as he walked, etc. etc.

earlier editions of this work, before we had these signs, I was able to say definitely that spastic paresis in its typical form and development does not occur in hysteria. On the other hand, it is certain, in spite of Babinski's[1] statement to the contrary, that the existence of ankle and patellar clonus is not conclusive evidence, as these symptoms are not uncommon in the neuroses and psychoses. Although the ankle clonus of the functional neuroses can usually be distinguished from true clonus, this is not always the case. My experience coincides with the statements of Bonhöffer[2] and A. Westphal.[3] Hysterical intention tremor is not so intimately connected with voluntary movement as that of sclerosis; its dependence upon mental processes is always distinctly evident. Moreover, it is inconstant and changeable in character. Scanning speech is almost always a late symptom in disseminated sclerosis; the allied speech disturbance of hysteria may be found at every stage. In such cases it is very marked at the onset, but is not uniform; some words are scanned or mangled in an irregular way, others are shot out rapidly. As disseminated sclerosis is often combined with hysteria, it may in some cases only be necessary to determine whether all the symptoms are hysterical or not.

One characteristic of hysterical symptoms is a trustworthy guide to their recognition, viz., *their dependence upon and their reaction to mental influence.* Every symptom must be examined in this light. The patient's attention should be distracted, and the effect upon his symptoms observed, his emotions aroused and their influence watched. When this does not succeed, another device which is often successful, especially in infantile hysteria, must be tried. We may assure the patient that pressure upon this or that part of the body will cause the symptoms to disappear or will elicit others. We should bear in mind the existence of hysterogenic zones, from which attacks are often excited or arrested. When this is not successful, it may be advisable to use *hypnotism*, which is often a great help in diagnosis. A suggestion that the patient should close his eyes and go to sleep is often sufficient to cause the most marked symptoms—the violent tremor, contracture, etc.—to disappear. As regards Westphal's pseudo-sclerosis, see p. 343.

Cerebral tumour and cerebral syphilis can only be mistaken for hysteria when ophthalmoscopic examination shows no change in the fundus oculi and when the symptoms are merely subjective. The diagnosis can in most cases be inferred from the kind of headache and the mental condition of the patient. Hysterical headache may certainly be very severe and violent, causing the patient to cry out, scream, and gesticulate wildly, while the headache of tumour, as a rule, renders the patient dull and usually stuporous, and betrays itself in the facial expression, although the patient does not complain of it. This is naturally not an absolute rule. The pulse is often slow at the height of the attack. Vomiting is only observed as an accessory symptom of hysterical headache when it has the character of hemicrania.

There may in rare cases be difficulty in distinguishing hysteria from paralytic dementia. I have detected hysteria in a case which an eminent alienist had diagnosed as paralysis. The deciding criteria need not be specially discussed. See Joffroy, *Bull. méd.*, 1904.

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[1] *Gaz. méd. des hôp.*, 1900; *R. n.*, 1903, see p. 235, etc.

[2] *Psych. Abh.*, edited by Wernicke, 1896.

[3] *N. C.*, 1903. For the diagnostic criteria afforded by graphic studies, see Claude-Rose, *R. n.*, 1906, and E. Levi, *Obersteiner*, 1907.

Hysterical paraplegia may be mistaken for *myelitis*. The bladder disturbance which sometimes accompanies it hardly ever takes the form of incontinence, but almost always of retention caused by spasm of the sphincter. A still more important symptom is that the sensory disturbance practically always exceeds the limits which would be found were they due to a focus of myelitis. It is as a rule associated with some sensory disturbance, and careful examination will disclose the peculiar psychical anomalies of hysteria. The presence of spastic or flaccid degenerative paralysis decides in favour of an organic disease. The differential diagnosis between hysteria and other diseases of the spinal cord, *e.g.* gliosis, has been considered under their respective headings.

Like Brodie, Paget, and others, I have also seen cases of hysteria which have been mistaken for *caries* of the cervical vertebræ, on account of the presence of rigidity of the neck, pain, girdle sensations, and motor disturbances. The sensitiveness of the vertebra to pressure was not, however, so circumscribed, and was generally discovered to be due to cutaneous hyperæsthesia; the contraction of the neck muscles was excessive, and finally the limits of the sensory disturbance surpassed those observed in cervical myelitis.

In one case in which the patient lay in bed for months, suspended by a Glisson's apparatus, I was able suddenly to cure all the symptoms through mental influence, and to allow the patient to run about in the garden on the same day.

The points of difference between *hysterical affections of the joints* and true joint affections have already been described. Many errors have been made regarding this. In one case under my observation, resection of the knee-joint had already been decided upon, and it was only upon the recommendation of a physician who was consulted that the patient was brought to me. She came on two crutches and left in a quarter of an hour without them.

In another case a shoe with a high sole had been worn on account of hysterical coxalgia with apparent shortening of the leg; the patient was able, after a few treatments with electricity, to dispense with it. In a third case a boy, after seeing a child suffering from bilateral club foot, acquired by imitation or mental infection an hysterical contracture of both feet with marked equino-varus position, which remained undiagnosed for a long time. He improved so much in a single treatment that the foot was held in its normal position.

In a few cases where laparotomy has been advised for visceral pain, I have been able to prove its psychogenic origin, and to bring about complete recovery or marked improvement by appropriate treatment.

Hysterical *neuralgia* may usually be recognised from its relationship to the hysterogenic zones, its causal dependence upon mental excitement, spasms, etc. Hysterical neuralgia is rarely confined to the area of a certain nerve, *e.g.* it is less often felt in some branches of the trigeminus than in one whole side of the body, and it often radiates into the shoulder, arm, and back. Pressure-points, like those of true neuralgia, are found, as well as hysterogenic zones, from which the attack can be provoked or arrested. The dependence of the various attacks upon mental excitement is also important.

Hysterical otalgia or mastoid pain has repeatedly been wrongly treated by operation.

In some cases the pain is the after-effect of a true pain, which has

continued after the cure of the original trouble in the form of psychalgia or habit pain (Brissaud's *douleur d'habitude*).

Hysterical neuralgia of the liver may simulate cholelithiasis. It is said that even jaundice may have this origin.

Böttiger's view that the so-called hysterical stigmata are artificial products, created by suggestion, is in our opinion an erroneous one, or at least one not of general application. Even were he right, these symptoms would retain their diagnostic value.

In some cases the hysterical or neuropathic disposition is revealed at the first glance by the look and attitude of the patient, by his excitability and restlessness, the haste of his speech and gesticulations, his extravagant speech, the rapid muscular tremors, like those of tic, etc. His expression is often somewhat characteristic. In many cases of neuropathy or psychopathy, the eyes are widely open, *i.e.* the marked dilatation of the palpebral fissures, etc., which only occurs in healthy people during violent emotion, is produced by the slightest excitement in conversation, etc. This is probably a sympathetic phenomenon. In a few cases I have been able to evoke the symptom through fright. On the other hand I have often seen hysterics raise the eyebrows (by means of the frontales) while the eyelids are lowered, as if it were a trouble to lift the eyelids, or as if there were a struggle between the orbicularis oculi and the levator palpebræ superioris which the frontalis had to decide. Naturally little weight can be attached to these indefinite signs.

We cannot sufficiently emphasise the necessity for the greatest care and thoroughness in deciding what importance to attach to the pain, even when the patient is hysterical. Pain should never be termed hysterical or psychogenic until a careful examination has excluded the possibility of any *organic disease of the bones and joints*, of a tumour of the nerve or in its neighbourhood, a constitutional disease, etc. *The neurologist who may easily err in making a differential diagnosis between an organic and functional nervous disease, should never for a moment forget that some other organic disease may be the cause of the symptoms. The errors of this kind, of which I have myself been guilty, though fortunately seldom and without grave results for the patient, have made me resolve to be silent concerning the errors of others which have come under my observation.*

I shall give one case as an example. A doctor brought his sister to consult me; she had complained for a year of pain in one thigh. As there were at first no objective symptoms, she had been regarded as hysterial and had been urged by him to walk. In climbing the steps leading to my house, she fell down, and I found that there was a spontaneous fracture of the thigh resulting from a malignant tumour of the bone.

The differentiation may be very difficult when an individual who is undoubtedly hysterical is suffering from an organic disease. Even the most experienced physician may make a mistake, if, *e.g.*, a malignant tumour (usually a mammary carcinoma) has been removed from an hysterical (hypochondriacal, etc.) woman, and she is watching, expecting or dreading a relapse or a metastasis. Metastatic tumour of the spinal column or the brain may at first cause symptoms which absolutely simulate hysteria and are therefore for a long time unrecognised.

We must also bear in mind the frequent combination of hysteria with organic diseases of the central nervous system (Charcot, Oppenheim,[1] Schüller, Hoppe, etc.).

Hysterical angina pectoris may resemble the true form. The pain usually commences suddenly; it is from the first very severe and radiates

[1] *N. C.*, 1890.

not only into the left arm (especially the ulnar region), but sometimes over the whole of the left side or into both arms. The skin may be hyperæsthetic. The pulse is normal or accelerated, but it has been observed to be intermittent. There is usually anxiety and a feeling of faintness, and there may be confusion. The comparative youth of the patient, the evidence of psychogenic origin, and the influence of hysterogenic zones are points of great importance.

Broadbent (*Lancet*, 1905) emphasises the fact that true angina is always caused by physical overstrain.

Hysterical clavus has been mistaken for acute meningitis, as it may be associated with vomiting, opisthotonus, and even with slight retardation of the pulse. The rapid, non-febrile course—a slight rise of temperature has been noted, but only in very rare cases—the presence of hysterogenic zones, and other symptoms of hysteria, together with the absence of paralysis of the cranial nerves, practically always prevent such an error. However " pseudo-meningitis hysterica " is very often mentioned, especially in French literature ; indeed, lumbar puncture has been resorted to in some cases of this kind. It has already been shown on p. 761 that the differential diagnosis may be very difficult.

The chapter on epilepsy should be consulted as regards the diagnosis between *hysteria* and *epilepsy*.

It is not necessary to describe here the points of difference between hysteria and *strychnine poisoning*, although they have been mistaken for each other.

Catalepsy is usually a symptom of hysteria, but it may occur in other psychoses, and in uræmia, etc., as Brissaud,[1] Biot, and Bauer,[2] among others, have shown. Prolonged cataleptic conditions in " undefined " attacks are practically always hysterical in nature (Lasegue, Binswanger). Spasms closely resembling *tetany* may be due to hysteria. This has frequently been observed by Schlesinger, Blazicek, A. Westphal, Curschmann,[3] Funcke,[4] myself, and others, but the characteristic signs of tetany, notably increase in the electrical excitability, are absent.

So-called hysterical *chorea* is usually of a rhythmic character. True chorea may also be combined with hysteria, or after the chorea has disappeared hysteria may produce a remission very similar to chorea, and in such a case the hysterical nature is not always easy to discover. So-called *chorea magna* is identical with the severe convulsive attacks of hysteria, and has no connection with chorea.

Adipositas dolorosa (*q.v.*) may be associated with subjective symptoms closely akin to those of hysteria.

Charcot refused to adopt the term hystero-epilepsy, as the convulsions never blend with each other, and there is no transition form. The condition formerly regarded as hystero-epilepsy is in reality hysteria. Epilepsy and hysteria may, however, co-exist as independent diseases. According to my experience, some cases undoubtedly show attacks both of the hysterical and the epileptic types. We cannot, however, deny the possible existence of convulsive conditions, which seem to represent a transition between the two. I[5] have specially observed these *intermediate convulsions*, which did not strictly correspond either to the epileptic or to

[1] *Progrès méd.*, 1903. [2] *R. n.*, 1903. [3] *Z. f. N.*, xxviii.
[4] *Prag. m. W.*, 1903. [5] *B. k. W.*, 1903 ; *Journ. f. P.*, vi.

the hysterical condition, in persons showing symptoms of the psychopathic disposition, in neurasthenics, and psychasthenics. Binswanger has since made a similar statement. Mixed forms have been described by Jolly, Nonne,[1] and Steffens,[2] whilst Hoche [3] and Bratz-Falkenberg [4] do not believe in the existence of a hystero-epilepsy. Bratz has since, however, come more to our opinion. (See chapter on epilepsy.)

See also Ziehen, " Psychopath. Konstit.," *Charité-Annalen*, xxix.

The diagnosis of hysteria from neurasthenia is discussed in the following chapter. These neuroses are very often combined. Hypochondria and the psychopathic conditions of fixed ideas, etc., to be discussed later, may also be associated with hysteria. There are many cases therefore which cannot be strictly diagnosed as belonging to any one of these neuroses.

Dementia præcox in its initial stage has many points of resemblance with hysteria. As to the diagnosis between them, the text-books on psychiatry should be consulted, and Nissl [5] should be referred to with regard to the occurrence of hysterical symptoms in simple mental disturbances.

Hysterical vomiting will seldom be confused with gastric diseases, unless anorexia is present. A characteristic point is that it follows directly upon eating, and that the food is still undigested. The fact that pain is usually absent, and in doubtful cases an examination of the vomited matter, prevent the condition being taken for a toxic one (arsenical poisoning, etc.). The vomiting of pregnancy is very similar, but the pregnancy can be ascertained by the history or by examination. Many writers are inclined to regard the sickness of pregnancy as a symptom of hysteria. The gastro-intestinal symptoms of hysteria have been wrongly diagnosed as gastric ulcer, appendicitis, peritonitis, cholecystitis, etc., and have been treated by surgical operations.

Thus I have seen a case of severe hysteria and psychasthenia in which three of our most eminent physicians have diagnosed disease of the gall-bladder and recommended operation, with an entirely negative result.

See also Boas (*D. m. W.*, 1905), Korach (*Mitt. aus d. Grenzgeb.* xv.).

Appendicitis seems to be a frequent occurrence in nervous families (Schaumann,[6] Adler). Affections of the sensibility of the abdomen, especially hyperalgesia, are said to occur in appendicitis (Sherren, Pieser,[7] etc.). On the other hand I have found hypæsthesia, and even marked diminution of the abdominal reflex on the affected side, in visceral neuralgia.

As regards the differential diagnosis, the " periodic vomiting " of children, which may be associated with fever and the excretion of acetone, should also receive consideration (Misch, *Jahrb. f. Kind.*, 1905).

The greatest care and experience cannot always prevent mistakes being made, and there is no physician, however skilful, who has not been at some time led astray by hysteria. It is always better to overlook this condition than to diagnose it in mistake for some grave disease.

I should like specially to draw attention to the fact that in some persons there is a congenital—possibly due to some inhibition of development—or an early acquired weakness of certain segments of the central nervous system, *e.g.* the cerebellum, the medulla oblongata, or certain circum-

[1] *Mitt. aus d. Hamb. Staatskrank.*, viii.
[2] *A. f. P.*, Bd. xli.
[3] " Die Differentialdiagnose zwischen Hyst. u. Epil.," Berlin, 1902.
[4] *A. f. P.*, xxxviii.
[5] *C. f. N.*, 1902.
[6] *D. m. W.*, 1903.
[7] *M. m. W.*, 1903.

scribed areas (centres) of the latter. In such cases this part of the organism may at some time of the patient's life fail or become so exhausted that it becomes incapable, either temporarily or altogether, of fulfilling its functions. Disturbances then appear, which are not due to an organic disease nor of an hysterical nature, as they show no relationship to the sphere of the mental or emotional life. The functional disorders may then develop in repeated stages. Thus, to give an example, I have often found in the children of diabetics, a marked excitability of the bulbar (vasomotor, vomiting) centres, etc.

This view, discussed in the earlier editions of this work, has been since put forward, in a somewhat modified form, by other writers.

In a case described by Siemerling (*Charité-Annalen*, xv. xvii.), which differed in many ways from hysteria, there were congenital anomalies of development, *e.g.* a fissuring of the optic nerve.

The *prognosis* as to life is quite favourable. In some extremely rare cases death from anorexia has occurred during an attack of spasm of the larynx. A few hysterics commit suicide (Esquirol, Legrand du Saulle). Death was the result in two cases of intractable vomiting, in one of meteorism, and in another of exhaustion caused by general hyperidrosis. There is no actual proof of the statement recently made that in hysteria acute œdema may develop in the brain, as in other parts.

So-called *acute fatal hysteria*, a most rare disease, occupies a special place. It resembles acute delirium with maniacal excitement, convulsions, and fever, and ends fatally within a few days or weeks. Examination of the nervous system was said to yield negative results, but the new methods of cell staining have shown certain changes in the nerve-cells of the brain (Alzheimer, Popoff), to which, however, subsequent experience has shown to be of no importance.

We would refer to the interesting communication of Reichardt (*C. f. N.*, 1905) upon mental affections closely allied to hysteria, but not identical with it.

The prognosis of hysteria as to *recovery* is on the whole favourable as regards the individual symptoms, but is doubtful as to the disease as a whole.

Each one of the symptoms, however long it has existed, may suddenly disappear, either spontaneously or under the influence of treatment. The prospects of recovery are the better the more recent the disease, the less it has been treated, and the greater the patient's faith and trust, and his determination to recover. Hysterical symptoms which develop in childhood, can almost always be easily cured, and the prospects of recovery from the disease itself are more hopeful. This has been lately emphasised by H. Curschmann.

The general convulsions and rhythmic muscular tremors are often very persistent. The longer a contracture has been in existence, the more difficult is it to cure (Charcot). Somnambulism tends to disappear in adolescence or later, or to be replaced by other symptoms, but I know a few cases in which it has persisted until old age.

In order to cure the disease itself, we must succeed in removing its original cause. As it is so very often due to the patient's position in life, his uncongenial environment, domestic, social, pecuniary, and other

conditions, recovery is often brought about by changes in his surroundings and habits. I have known severe cases of hysteria completely cured by happy marriage, which removed the patient from a life of strain and anxiety, whilst an unhappy marriage is only too often the cause of hysteria. In other cases the realisation of anxious hopes, the cessation of anxiety as to the future, or the satisfaction of some morbid ambition has been the means of recovery. The persistence of the condition, however, makes the outlook in this direction less favourable.

We must also consider the *degree of hereditary predisposition*. If this is considerable, and is early revealed by signs of degeneration, stigmata and marked mental abnormalities, there is very little prospect of complete recovery. If, on the other hand, hysterical symptoms occur in an individual in whom one would never have expected them, and whose whole disposition seems opposed to them, the hope of recovery is much greater. Hysterical conditions which are produced by imitation are usually of short duration.

There is a class of cases (Ziemssen, Heyne, Strümpell, Siemerling, Oppenheim, Burr, Seifert,[1] etc.) in which the anæsthesia is total, and associated with abulia and ultimately with general paralysis of the body, so that the patient is permanently confined to bed. Anorexia usually develops, and finally hallucinatory paranoia. In a few of these cases death has taken place from inanition or some complicating disease. It is difficult to decide whether this condition should be regarded as hysterical. In one such case a post-mortem examination which I was able to make threw no further light on the condition. This case is described as Nr. xxiii. in my monograph on traumatic neuroses.

Hysteria in the male, especially when atypical and combined with neurasthenia, hypochondria and any psychosis, is specially intractable.

Treatment.—Suitable *prophylaxis* may do much good. The physician should specially impress upon the hysterical mother that she must not complain or speak of her illness in the presence of her children, or devote excessive attention to each childish complaint. When this precaution has no effect, one should urgently recommend that the child be removed from its family circle. This, of course, is only necessary when the nature and temperament of the child show a tendency to hysteria. Unfortunately those children are usually most sensitive ; they cling with exaggerated affection to their mother, and both mother and child protest strongly against separation. It is therefore advisable to recommend some treatment which cannot be carried out at home.

The child's *education* (see Oppenheim, " Nervenleiden und Erziehung," Berlin, 1899 ; 2nd. ed., 1907 ; Forel, " Hygiene der Nerven und des Geistes," etc. ; Biedert, " Das Kind," etc., Stuttgart, 1906, and Neter's excellent paper, " Das einzige Kind und seine Erziehung," München, 1906) should be strict and regular, but not harsh and rigorous. The child should not as a rule be treated with threats or intimidation, as this is a very two-edged method. Any tendency to sentimentality should be at once repressed. A strong effort should be made to teach the child from an early period to control his emotions.

This point should receive special attention in the treatment of nervous persons. It is possible for a sensible person, by scrupulous self-training, to maintain an equable temper, out of which he

[1] *Z. f. N.*, xxviii. See bibliography here.

cannot be easily roused. This requires stern self-restraint and the resolve to translate " mental excitement " into conscious action, sympathy into help, despair into activity which will tranquillise or at least ease the mind, etc. The feeling of anger, in particular, should be immediately repressed by the remonstrance which a nervous person should always be prepared to address to himself, and by conscious activities which will absorb his attention.

Everything that over-excites the child's mind should be avoided. His reading should be supervised, and he should be kept as long as possible from theatres and concerts. On the other hand he should be as much as possible in the open air, gaining physical strength by playing, cycling, swimming, riding, rowing, etc., and avoiding all mental strain. A child with a neuropathic disposition should be early taught to be *observant of external objects*, to occupy himself with the outer world (the study of nature, of technical and geographical subjects, of farming, etc.), and his altruistic interests should be cultivated to the utmost extent.

If the disease has developed during childhood, the best method of treatment is to *remove the child from its family circle*, and if that is not sufficient, to place him in a *suitable institution*. This is often the only proper course. I have frequently observed that hysterical boys who suffer from hallucinatory delirium, are cured as soon as they are placed in a different environment. In a few cases the whole mental training was entrusted to the family of a schoolmaster, clergyman, etc., with excellent results. In many of these cases it is advisable to send the child to a home in the country, but this requires to be carefully selected.

The first duty of the physician is to discover the origin of the disease and then to try to remove the cause. It may be due to physical conditions, which require to be treated with iron or strengthening food. The pallor of nervous people is, of course, often not a sign of anæmia, but of vascular spasm, and in this case it does not respond to iron, but to some other form of treatment (general or mental). Even chlorosis has of late been attributed to a primary disease of the nervous system, especially of the vaso-motor system (Grawitz). The iron waters of Pyrmont, Elster, Schwalbach, Rippoldsau, Kainzenbad, St Moritz, etc., may, however, be of real service in the treatment of this disease.

The *diet* should be nourishing and should consist of simple, ordinary, easily digested food, especially milk. If milk is not well borne, kephir or some other substitute should be tried, or lime-water or soda added to the milk. Alcohol in any form should be forbidden or strictly limited to a small quantity. Hysterical women are sometimes in the habit of taking rather large quantities of wine or brandy. Coffee and tea need not be so strictly forbidden as some physicians think necessary, as they are only harmful when taken to excess.

It may be very difficult to lay down a line of treatment when there is also some *disease of the genital system*. If gynæcological treatment is likely to be prolonged and uncertain in its result, it should be generally avoided. But if the trouble is slight and is likely to be cured by a single operation, it should be recommended. (Narcosis with chloroform and nitrous oxide may entail some risk in hysterical cases.) The urgency of the indications must of course decide the procedure in each case. Above all, one need never hesitate to perform the less serious gynæcological operations, which can be carried out at one or two occasions, and which neither weaken nor excite the patient. Such operations have lately been recommended from personal experience by American and German gynæcologists

(Sherwood-Dunn, Krönig, Theilhaber, etc.). Ovariotomy has seldom a good effect, especially in chronic cases. Naturally it should only be undertaken if indicated by the ovarian disease itself. We should remember that amenorrhœa is often a *symptom of nervous disease*, and that hæmorrhage may also be due to this cause. Profuse menstruation may be improved by plugging (Kussmaul, Klemperer[1]). The application of cocaine to the mucous membrane of the nose may cure *dysmenorrhœic* symptoms (Fliess, Schiff, etc.). I have found ovarian tablets useful in many cases of climacteric disorders (Landau). Gottschalk highly recommends hot baths (32-33°, for twenty minutes) for the congestive symptoms of the climacteric. Carbonic acid baths may be beneficial.

It is seldom necessary to bring about abortion, but this necessity may be indicated by severe and dangerous impairment of nutrition due to excessive vomiting or great depression with suicidal impulses, etc., caused by the hysteria. A discussion published in the Hamburger *Naturforscherversammlung*, for 1901, shows how widely opinions still differ on this subject.

See also Gross and Wagner-Jauregg, *W. kl. W.*, 1905; Pick, *ibid.*; Friedmann, *D. m. W.*, 1908. The old rule that hysterical women should be advised to marry, has from the experiences quoted above and the views developed by Briquet, Pitres, and Voss, been abandoned, although it must be admitted that a *happy* marriage may have a very beneficial effect.

A change of environment is one of the most important elements in treatment of the hysteria of adults. Most of the mental excitement which has been so harmful to the patient may thus be removed. Residence in the country, at the seaside, or in the hills may be good for this reason. Sea-bathing is often badly borne by hysterics. *Treatment in a nursing-home* or institution for nervous diseases, where a careful physician, trained in neurology and psychiatry, can watch the entire life and habits and acquire a complete understanding of the mental condition of the patient, is most likely to be successful. Unfortunately there are not many such institutions which answer all the requirements in the way of management, nursing, comfort, etc. If may be necessary under certain conditions to isolate the patient completely. This measure has been specially recommended by Dejerine,[2] who has in many cases isolated his patient in a dark room, with excellent results. We should like to know if these numerous recoveries have really proved permanent.

Work is a remedy of immense value. It must of course be adapted to the capacity and strength of the patient, and should be as varied as possible. The indications have been carefully discussed by O. Vogt and Veraguth.[3]

Institutions in which work is employed as a means of treatment have recently been started at various places, and this factor is taken into account in many sanatoriums. The advice given by Möbius has been specially helpful in this respect, and his recommendations have in part at least been carried out in some German institutions, *e.g.* the neurological institution at Zehlendorf. We might also refer to the child-gardens of Köstritz, Werneuchen, Marienfelde, Gera, Treffurt, etc., and to the agricultural institute of Dr Jacobi at Wetterscheidt (and Warnke's similar undertaking), which are specially adapted for the employment of mentally under-developed youths of

[1] *Therap. d. Geg.*, 1903.
[2] *R. n.*, 1902. See also Camus et Pagniez, "Isolement et Psychothérapie," etc., Paris, 1904.
[3] *Therap. d. Geg.*, 1905. See also Laehr and Lachs, *Z. f. kl. M.*, Bd. liii., and Laehr, *A. f. P.*, xl., with regard to the cases treated in Haus Schönow.

the better classes. Dr Heimann (Cauerstrasse) has built an institution in Berlin where nervous patients are employed for hours at a time in gardening.

Some of the *general methods of treatment* are intended to have a strengthening, stimulating, and diverting effect upon the nervous system (consult the indications laid down in the chapter on neurasthenia). These include in particular *hydropathic*, *climatological*, *medico-mechanical*, and *electrical* measures. The result can never be guaranteed in any particular case. So many factors are involved, and the result depends so greatly upon the faith of the patient, that treatment must always be to a certain extent experimental.

Psychotherapy is the essential factor in the treatment. The physician must bring an intense and warm interest to bear upon his patient, whose confidence he must gain without losing the prestige of his authority. He should speak as man to man, and his influence may at first be prejudiced if he is hampered by having to remember the qualifications of title, rank, and position. The physician who is dealing with the mind should be allowed the greatest possible freedom from these formalities, and should address his patient directly as "you." It may be particularly difficult to adopt the right tone and attitude when the patient is a woman—to show on the one hand sufficient sympathy and on the other to avoid cordiality and the rousing of erotic tendencies. The physician should not ignore any of his patient's troubles; still less should he ridicule and laugh at them. He should constantly repeat his conviction that they are part of a curable condition, and constantly hold out a definite prospect of recovery. He must teach the patient how to train himself properly, and convince him that great results can be attained by the distraction of his attention from his symptoms and by the gradual strengthening of his will-power. Understanding and tact on the part of the physician will gain the trust of the patient, who will lay bare to him his inner life and will confide to him his secret mental troubles, which must be removed if the disease is to be cured. On the other hand some unfortunate expression on the part of the doctor may cause untold harm; the opinion or even the suggestion that some organ is diseased makes an unalterable impression and remains firmly fixed in the patient's memory. Moreover, every physician is not suited to every patient. In some cases an iron bearing is necessary, any sign of gentleness or intimacy weakening the confidence of the patient. Hypnotism can generally be dispensed with. The method of Freud and Breuer, which consists in awakening, during a hypnotic condition or in a confidential talk, the processes which have entered into the mental life and caused the disease, has not been approved of by other writers, and recent personal experience has led me to agree emphatically with those (O. Vogt, Jolly), who regard this kind of treatment as dangerous. We can only refer to the modified method of "psychosynthesis" of Frank-Bezzola.[1] The principles of psychotherapy have been thoroughly developed by Ziehen, Vogt,[2] Oppenheim,[3] Loewenfeld,[4] Hartenberg, Dubois,[5]

[1] *N. C.*, 1906; *Journ. f. P.*, viii. [2] *Journ. f. P.*, i., etc.

[3] "Nervenleiden und Erziehung," etc.; also "Prognose und Therapie d. schweren Neurosen," Samml. zwangl. Abhandl., Halle, 1902; and "Psychotherap. Briefe," Berlin, 1906. English trans. by A. Bruce, Edinburgh, 1907.

[4] "Path. und Therap. der Neurasth. und Hysterie," Wiesbaden, 1893.

[5] "Die Psychoneurosen und ihre psych. Behandlung," Bern, 1905.

Lévi,[1] Renterghem,[2] and others. We would in particular draw attention to the excellent treatise of Dubois, although his views and teaching are somewhat one-sided. Among the older contributions, that of Rosenberg [3] should be remembered.

It need not be said that the physician should never be led into chastising a hysteric, a procedure which moreover is illegal.

It is often the physician's task to combat *individual symptoms* of hysteria, which cause special distress to the patient. The majority of our remedies have no direct effect, but are successful when they convey to the patient the idea of their power to cure him. They therefore usually lose their influence in time, and new methods must constantly be tried to produce the mental influence which we desire. If the family physician has exhausted all his resources, a specialist may be called in, who may effect a cure by prescribing some remedy which in itself may be indifferent. A simple command or the assurance "you can" is often sufficient to overcome the hysterical symptom in children. *Hypnotic* treatment is justified in very obstinate cases. We have no right to put any obstacle in the way of a credulous patient who seeks relief from some miraculous spring.

In addition to psychotherapy and the general treatment already discussed, other methods which may have a reflex, circulatory, or other effect upon the nervous system should be employed. These include treatment with the *faradic brush*, *static sparks*, *cold douches and baths*, and other hydrotherapeutic or bathing methods (carbonic acid baths, friction with carbonic acid, pine-needle baths, etc.), counter-irritants, *massage*,[4] and *gymnastics*, vibration massage, sun and light baths. The method of *cultivating self-restraint* (Hemmungstherapie), which I have recommended, and which consists of systematically keeping the extended limbs at rest, and suppressing all emotional or reflex movements, has often proved very beneficial in hysterical conditions.

These methods apply more or less to all the forms and symptoms of hysteria. Let us now consider the more special symptoms.

The faradic brush is particularly good for the *anæsthesia*. This condition may also be cured by the application of metals, sinapisms, and magnets. A horse-shoe magnet of the largest possible size should be used. The electric sparks of the static machine have the same effect. It is often successful when other methods have failed.

For the *headache*, backache, neuralgia, etc., the constant current should be applied to the site of the pain. Hydrotherapeutic methods, such as the application of an ice-bag or a Priesnitz's pack for pain in the back, are often valuable remedies. *Counter-irritation* to other parts, *e.g.*

[1] *Presse méd.*, 1903. See also Goldscheider's article on "Die Stimmung" in *Der klin. therap. Woch.*, 1905, and *Z. f. diät. Ther.*, 1906.

[2] "La Psychothérapie," Amsterdam, 1907.

[3] "Nervöse Zustände und ihre psych. Behandlung," second edition, 1903.

[4] In massage the mental factor may also play its part, the method being thus not a purely mechanical one, but a psycho-manual action. Even in healthy persons, contact, the touch of another person's hand may evoke psychologically different sensations. A clear proof of the intimate relation between the muscular action of the hand and the life of the mind is furnished by the effect of music played by a musician; his technique apart, it is his emotion which is translated into movement, and the magical effect produced is therefore the result of a psycho-motor act. It is very apparent, for instance, that the mental hand of a Joachim differs in this respect from the hand of any other man. This fact explains why the individual factor plays so great a part in massage and other manipulations of this kind. And how much greater is this influence in the case of nervous subjects!

the faradic brush to the soles of the feet or a foot-bath, may have a marked effect. The static breeze applied to the head or the back, the Arsonval, and magneto-electric currents may also be used.

"Hysterical pseudo-meningitis" has even led to the use of lumbar puncture.

I have attempted to combat the psychalgias by systematically exercising the patient in the disregard of irritations which arise from the painful parts of the body.

To give an example, I hold a watch to the patient's ear, so that its ticking absorbs his whole attention, to such a degree that he does not feel any touch upon the painful part. Or I touch simultaneously two parts of the body, one in the region of the focus of pain, the other at some distant point on the skin. By making the latter stimulation the stronger or by using some specially fine stimulus at this part, which the patient has to localise, it can easily be contrived that the simultaneous touch upon the affected part is not perceived. By persistent exercises of this kind one may succeed so far that when two stimuli of equal strength are used, that applied to the distant site will alone be consciously perceived. I then proceed to use painful stimuli, such as the prick of a pin, pinching a fold of skin, etc., and by increasing the strength of the distracting stimulus I contrive that it alone will be consciously felt. Finally, one will reach a stage in which the patient, if he will, either succeeds in ignoring painful manipulations on the affected part, or does not feel them to be painful. He is thus trained not to direct his attention in a pathologically exaggerated degree to the site of the pain. Of late I have mostly used the faradic brush to produce the distracting stimulus.

Massage, vibratory-massage, brushing the skin, and similar mechanical irritations may be of use for hysterical pain.

As to *drugs*, it is always advisable to begin with indifferent and mild remedies, such as *valerian*, *validol*, *valyl*, *bornyval*, asafœtida, the bromides in small doses (7½-15 grs. of potassium or sodium bromide several times a day), *quinine-hydrobromide* (2-3 grains in the form of powder or pills), bromalin, bromipin, etc. Should these drugs fail, the *antineuralgics* may be tried. The patient's imagination may be impressed by the astonishing effect of methylene-blue, as the cures reported by Pitres show, but I have not often obtained this good effect.

Narcotics proper (morphia and chloral) may almost always be avoided. Any harmless remedy may be used as a means of mental treatment. I have often succeeded in inducing sleep by prescribing *pulvis gummosus* or sacch. alb. after all the other drugs had failed. Gargling with a solution of bromide, chloride of sodium, etc., should be prescribed for the *globus*. Should this fail, an emetic may sometimes be successful, or the use of the faradic current, one electrode being introduced into the pharynx. This may also be employed as a remedy for intractable hysterical eructation. In one such case, in which double ovariotomy had been performed without any result, I obtained complete recovery by suggestive treatment. Dysphagia may also sometimes be cured by a single introduction of the œsophageal sound.

The *attacks* often resist every kind of treatment. Bromides are much less effective than in epilepsy. If the spasm can be elicited from a cutaneous hysterogenic zone this should be protected from contact by a pad; if the zone lies in the deep parts or the internal organs, some counter-irritant (cantharides, button cautery) may cure the condition.

If the attack has already commenced, sprinkling with cold water or repeated cold douches may have a sedative influence, or it may be arrested by pressure on the ovarian region. Pressure on the region of the apex beat

of the heart less often has this effect. There is often nothing to do but to allow the attack to exhaust itself, to protect the patient from injury, and to avoid paying too much attention to him. He should be isolated and left to himself. Judicious neglect (Fürstner) may be a very effectual remedy. Reasoning and forcible fixation of the extremities are of no use ; on the contrary, they usually increase the intensity of the spasm. In some very severe cases, I was able instantly to stop the attack by suddenly presenting a magnet to the patient. Loewenfeld recommends *hyoscin*. Quinine (10-15 grains an hour before the attack), pilocarpin, and duboisin are recommended for prevention of the attack, but these remedies are of very little value.

Counter-irritants should be used for local spasms ; if these are not successful in cases of spasm of the glottis, an emetic (apomorphin given subcutaneously) should be tried. Hiccough (singultus) may sometimes be arrested by rhythmic traction on the tongue (Laborde, Noir). Wolfberg recommends that in blepharospasm the healthy eye should be kept closed for some hours.

Huyghe reports having cured (?) hysterical chorea by fixing the limbs under chloroform.

In cases of contracture one should endeavour as early as possible to prevent the permanent development of muscular atrophy. Massage in combination with psychotherapy is often a means of cure. Tight bandages, especially plaster of Paris dressings, should be entirely avoided. The use of the constant current, the magnet, or the static breeze may be tried. Should these have no effect, the contracture may sometimes be cured by inducing, if possible, an attack of spasm. The contracture not infrequently disappears as the spasm passes off. In one case of hysterical curvature of the spinal column, epidural injection of cocaine was said to have effected an immediate cure (Deléarde).

Lumbar anæsthesia or the muscular relaxation which it causes has been said to cure hysterical contracture (Wilms, *D. m. W.*, 1906 ; Löhrer, *M. m. W.*, 1906).

The paralytic condition most often met with in practice is *aphonia*. In recent cases the use of the laryngoscope or of a sound, some indifferent drug, etc., pressure upon the throat, etc., is often sufficient to bring back the voice. Should these methods fail, we may use the faradic current (in the form of the brush or of muscular stimulation). Franklinisation may also be recommended. If these remedies fail, intralaryngeal stimulation of the muscles of the vocal cord is often successful. Cutaneous massage, compression of the larynx, a kind of respiratory gymnastics—the patient being made first to expire forcibly, then to cough, then to produce a sound along with the cough, and finally to blurt out a word as he coughs, etc.—and hypnosis may also cure the condition. A sounding tuning-fork placed upon the breast at the same time as the patient makes an effort to phonate has also been recommended (Maljuto). In one very obstinate case under my care the voice returned when I made a small incision in the arm of the patient without the use of an anæsthetic ; she fainted and thereafter recovered her voice. In another case in which every method had been tried in vain, I applied a *seton* to the neck and thus induced permanent recovery.

If the paralysis affects the limbs, it is advisable to urge the patient to perform active movements and to supplement these at the same time

by passive movements under continual suggestion. The sensations of movement help to divert the current of innervation into the paralysed muscles.

In hysterical *paraplegia*, walking should be tried as soon as possible, at first carefully, the patient being well supported, until he learns to move alone, resting his arms upon some firm support. Recovery as a rule follows very quickly. In such cases I have found walking exercises between parallel bars specially useful. A " go-cart " is not necessary. If there is a tendency for the knees to give way I fix them lightly with a few turns of a bandage. This method is preferable to the instantaneous recovery or " surprise-method," in which all the symptoms are completely banished in one sitting. If all these methods fail, we may be justified in invoking an attack of spasm, which in many cases causes the paralysis to disappear (Charcot). It may also be advisable to chloroform the patient and to set him upon his legs before he fully recovers consciousness.

One must always remember that recovery from one symptom does not imply a cure of the disease, and that a relapse is not only possible, but probable, if the causal disease is not removed.

Hysterical vomiting often resists all treatment. Valerian, bromide, quinine, chloroform (2 to 5 drops in mucilage), etc., may be prescribed, and chloral hydrate (1 : 50, 10 to 20 drops), has been specially recommended for cardialgia (Rosenbach, Ewald). When this is not successful, it is advisable to order a simple consistent diet, such as raw ham or scraped meat, or to feed the patient by means of an œsophageal sound or enemata. In intractable cases one may resort to an emetic or to washing out the stomach. " Internal massage " of the stomach by means of the œsophageal sound has also been recommended (Richter), but this is not a necessary procedure. Anorexia is a very persistent symptom, but it is often cured by the use of the œsophageal sound. In many cases which resist other measures, Weir-Mitchell treatment has effected a cure (see following chapter).

Constipation seldom yields to the ordinary laxatives. Purgatives should not be given. If small doses of a drug produce no effect, larger doses are not likely to do so. Treatment at watering-places, such as Marienbad, Homburg, etc., is not usually beneficial. An hysterical lady under my care discovered that her bowels acted whenever she administered castor-oil to one of her children, although this and other drugs had no effect when she took them herself. The uselessness of drastic remedies in hysteria could not be more convincingly demonstrated. *Cold-water enemata* are more suitable, and *electrical treatment* in the form of faradisation of the abdomen, galvano-faradisation with the use of a massage roller, intra-rectal electrical treatment, is particularly good in this form of constipation. Massage has often an excellent effect. The patient should form the habit of making his bowels act at a certain time, even should the impulse be absent.

For spastic constipation, local application of warmth (thermophor, hot irrigations) and the use of the Fleiner-Kussmaul oil-enema, etc., should be tried. These measures may also be employed in so-called nervous ileus, although this, like every other hysterical symptom, chiefly requires psychotherapy. Washing out of the stomach, or the introduction of an intestinal tube during narcosis, has occasionally been beneficial, but laparotomy has been resorted to in not a few cases.

APPENDIX

Hynotism and Hypnosis

Literature in the monographs of Forel, Moll, Hirschlaff, Vogt, the papers in the *Z. f. Hypnotismus*, etc.

The hypnotic condition is seemingly allied to that of sleep, but differs from it mainly in the mental bond (the *rapport*) which unites the hypnotiser to his subject. The former has the power of acting upon the imagination of the patient, of awakening ideas in his mind which under certain conditions become his mental property, and to a certain degree influence his physical functions.

Vogt starts from the conception of suggestion (compare the preceding chapter, p. 1058) as a psycho-physical phenomenon which represents an abnormally intense reaction to purposive ideas.

A purposive idea (" Zielvorstellung ") is the idea of a psychophysical process carried into action. He distinguishes between ideas of this kind, which rouse strong emotions, and those which rouse weak emotions, and characterises as *hypnotic* the condition of consciousness in which the suggestions which arouse the weaker emotions are responded to. Some writers who have carefully studied this question (Liébault, Bernheim, etc.) give the proportion of hypnotisable people as eighty per cent. : there is much doubt, however, as to the accuracy of this estimate.

Charcot and his pupils, who studied the hypnotic condition in patients with grave hysteria, thought it possible to distinguish three different stages which blended into each other :—

1. *The cataleptic condition.*—The eyes are open, the expression staring, and the body resembles a statue. The limbs can be placed in any position and remain in it without any sign of fatigue. The tendon reflexes are said to be absent (?) or diminished. Sensation is abolished, but the sensory functions are only partially impaired.

The cataleptic condition is produced by a sudden sensory stimulation (a sound, a bright light) or by making the subject fix his eyes upon an object.

2. *The lethargic condition.*—The eyes are partly or entirely closed, the muscles are relaxed, sensation and the function of the special senses are abolished. In this condition the patient is not open to suggestion. There is neuro-muscular hyper-excitability ; the muscles can be made to contract by tapping or percussing their nerves. The lethargic condition is said to be induced by pressure on the eyeball, or to be evolved from the cataleptic condition by shutting the eyelids.

3. *The somnambulistic condition.*—This may be directly induced by fixing the eyes upon a point by the action of weak and uniform stimuli to the senses (listening to a tuning-fork), or it may be evolved from the other phases by pressure on the crown of the head. The eyes may remain shut or open. The skin is insensitive to pain, while the special senses are active or even exaggerated. There is no neuro-muscular excitability, but muscular contractions can be elicited by mechanical irritation of the skin. This stage is chiefly characterised by the increased susceptibility of the individual to suggestion.

There is no longer any doubt that these three stages cannot be thus practically distinguished and characterised, and although all the symptoms described are occasionally observed, they are entirely the products of suggestion, and by no means necessarily belong to the condition of hypnosis.

A knowledge of these stages is only of interest in relation to corresponding symptoms which occur spontaneously in hysteria. It is of no practical value as regards hypnosis itself.

It is of course the case that hypnosis may gradually become deeper and may pass from a condition of the lightest sleep, in which spontaneity is almost completely retained, to one of unconsciousness resembling somnambulism. In practice, however, it is usually neither necessary nor advisable to push the hypnosis to such an extent.

Method of hypnotisation.—Hypnosis should only be practised by a physician who has faith in his own power, who has mastered the technique, and can devote himself with patience and perseverance to the treatment.

Children and mentally deficient persons need not as a rule have any previous explanations made to them. Intelligent adults must previously be instructed to repress as far as possible all opposing and incidental thoughts, and to think only of going to sleep, or of the ideas which the physician suggests to them. The physician must be calm and confident and possessed of the entire trust of his patient. The patient should sit in a comfortable position, the physician standing before him. He should then be asked to fix his eyes upon some object held before him—a shining button is generally used, but a finger will do—or to look straight into the physician's eyes. Concentration of the mental functions upon a given point is an important element in inducing sleep.

Additional measures may be employed, such as the so-called mesmeric passes, the palm of the hand being slowly and gently stroked in one direction over the face and eyes, or at a short distance from them. These accessory measures are usually not required, "*verbal suggestion*" being sufficient. The essential point is to call up in the patient's mind the idea of sleeping. If his *attention is concentrated upon this idea* the avenues of his senses are closed, his will slumbers, his thoughts are restrained, and the mental condition of increased suggestibility is thus brought about.

One should therefore say in a tone of conviction: "You will soon fall asleep, your eyelids already feel heavy, you can hardly keep them open," etc. If the eyes do not soon close of themselves, they may be gently shut by the physician, who says, as he does so, "Now you are asleep!" It is well, however, to explain to the patient that the sleep will not be complete, and that he will hear all that is said to him.

The first attempt may not succeed, or the patient may only feel his limbs slightly heavy. This indicates that he is susceptible, and further attempts will as a rule evoke the condition of increased suggestibility. He will then sit with his eyes closed, as still as if asleep, and if his limbs are passively raised they fall flaccidly back or remain raised at command (Fig. 410), etc. It is well known that the motor and sensory functions, the sensory impressions, and the whole mental state may be influenced in these conditions. We need not therefore discuss this in detail.

When this condition of hypnotism is attained, the idea should be suggested to the patient that his symptoms—the spasm, paralysis, contracture, anæsthesia, pain, etc.—have disappeared or will do so. It is advisable not to attack all the symptoms at once, but to suggest the gradual disappearance of one after another.

FIG. 410.—A patient in a hypnotic trance. (Oppenheim.)

It has been recommended, especially by Wetterstrand, that the hypnotic sleep itself should be used as a remedy to tide the patient over certain troubles and even to prolong the sleep for hours and days.

Breuer and Freud have drawn attention to another indication for hypnosis. They have found that some of the symptoms of hysteria are referable to some mental trauma, which the patient has not been able to discharge from his mind by an outburst of emotion. If, while he is in a hypnotised condition, it is possible to bring his mental trouble to his consciousness in such a way that he will speak of it and lighten the burden on his mind by weeping, or by a passionate outburst, etc., a serious source of trouble may be removed. It is as if a foreign body had been removed from a wound. As already mentioned, grave objections have been urged against this measure, which has recently been modified by Freud himself, and still more by Jung, Frank, and Bezzola. The patient is wakened out of his hypnotic condition by a simple command or a breath of air upon him.

A few cases (Gräter, Hilger, Riklin, see *Journ. f. P.*, i.) point to the possibility of clearing up epileptic amnesias by hypnosis, but it seems to us very doubtful whether this would be the case in true epilepsy.

I have thought it necessary to indicate the methods of hypnotisation, as I think it may occasionally be used with benefit. It is specially useful in the treatment of *psychogenic pain*, *paralysis*, *anæsthesia*, *spasm*, and *contracture*, vomiting and constipation. Its value is shown notably by the communications of Forel, Bernheim, Wetterstrand, O. Vogt, Loewenfeld, Delius, Renterghem, and others. Kohnstamm, who has published observations upon the influence of hypnotism upon menstruation, has made it easier for us to understand this mental influence upon the visceral functions by showing that they may be called into active exercise by awakening the corresponding sensory processes (*Journ. f. P.*, vii.; *Therap. d. Geg.*, 1907). Insomnia in hysteria or neurasthenia may be an indication for hypnotic treatment which Vogt and others have often employed with success. But it is always advisable to try the other therapeutic methods first, and to combine these with psychotherapy—*waking suggestion*. Children are almost always cured in this way. It is only when these methods fail that hypnosis should be employed.

This treatment also seems to me suitable for some cases of imperative ideas, and even more for conditions of anxiety. If ideas of a morbid nature occasion great distress to the patient or

injure his health, we are justified in directly attacking the morbid thoughts and, as far as lies in our power, in substituting healthy ideas for the morbid ones. This of course is impossible in the hallucinations of insanity, and it is generally a failure when applied to the true *idées fixes* (*q.v.*).

On the other hand it has been rightly pointed out that hypnosis may have a harmful effect and may produce symptoms of grave hysteria. It requires therefore to be used with care, conscientiousness, and thorough experience.

In doubtful cases hypnosis may be used to establish the diagnosis, as we have already shown in the previous chapter. O. Vogt has also shown that certain forms and degrees of the hypnotic condition (" partial systematic wakefulness ") are specially adapted to the investigation of psychological processes.

Neurasthenia or Nervous Weakness

Literature: Beard, " A Practical Treatise on Nervous Exhaustion," 1880 (German translation, 3rd edition, 1889); Möbius, " Die Nervosität," Leipzig, 1882; Axenfeld, " Traité des Nevroses," Paris, 1883; Beard-Rockwell, " Die sexuelle Neurasthenie," Vienna, 1885; Weir-Mitchell, " The Treatment of Certain Forms of Neurasthenia and Hysteria " (German, Berlin, 1886); Loewenfeld, " Die mod. Behandl. der Nervenschwäche," etc., Wiesbaden, 1889; *ibid.*, " Path. und Therap. d. Neurasth. und Hyst.," Wiesbaden, 1894; *ibid.*, " Sexualleben und Nervenleiden," 4th edition, Wiesbaden, 1906; Bouveret, " La Neurasthénie," Paris, 1891; Levillain, Paris, 1891; Binswanger, " Pathol. und Therap. d. Neurasth.," Jena 1896; Krafft-Ebing, " Nevrosität," etc., Nothnagel's " Handbuch," xii.; *ibid.*, " Psychopathia sexualis," 9th edition, Stuttgart, 1894; Oppenheim, in the earlier editions of this book; *ibid.*, " Die ersten Zeichen der Nervosität des Kindesalters," Berlin, 1903, 2nd edition, 1907; Martius, " Pathogenese innerer Krankheiten," iii., Funktionelle Neurosen, Leipzig-Wien, 1903; Cramer, " Die Nervosität," Jena, 1906; Savill, " Neurasthenia," 3rd edition, London; Ziehen, article on Neurasthenia in Eulenburg's " Realenzyklopädie "; Raymond, " Névroses et Psychoses," Paris, 1907.

Neurasthenia is a very common disease in our time. It affects chiefly those who live in large towns. Although it has possibly been in existence for all time,[1] and has for long been known under the term " nervousness," there is no doubt that it has gained ground enormously within recent years, with the ever-growing hurry and restlessness of social life, and the enormously increased demands made by the struggle for existence and the craving for pleasure. Difficulty in diagnosing hemiasthenia undoubtedly arises from the fact that no sharp distinction can be drawn between the slightest degrees of this condition and certain symptoms which may be looked upon as physiological, as even healthy persons may experience transient symptoms which, if strongly marked and persistent, can only be regarded as neurasthenic.

Neurasthenia occurs in both sexes, but in its pure form it is more common in men. It is not associated with any special age. Although it is mainly a disease of adult life, it is not unusual in young people and children of five to ten years of age, and it even may appear in early childhood. I have seen it in a severe form in a child of two-and-a-half, and have treated a large number of cases, greatly increased within the last few years, of children from three to five years old. Old people may also become neurasthenic.

Heredity is the most important *cause*. Slight causes are sufficient to give rise to the development of neurasthenia when there is an inherited neuropathic predisposition. There is also a *congenital* form of neurasthenia which has its first commencements in earliest childhood.

[1] Martius has shown that, even in 1843, G. Hirsch described the nature of this disease in a way which corresponds entirely with our modern views.

In such cases signs of physical and mental degeneration are comparatively frequent. Ziehen found similar heredity in 40 per cent. of his cases. A *toxicopathic* defect (alcoholism in the parents) may lay the foundation for neurasthenia in the children.

Emotional disturbances are prominent amongst the other causes. Neurasthenia may be directly caused by a single mental shock, but it is more frequently the result of prolonged or repeated emotional excitement. *Mental exhaustion* is also an element in the etiology. It has been rightly said that brain-workers are much more apt to become neurasthenic than those who work with their hands. *Over-work at school* may sow the seeds of the trouble. Quiet, regular mental work is not so harmful to the nervous system as hurried mental activity, which absorbs the hours of sleep and is associated with excitement of many kinds, anxious fears and hopes, painful disappointments, etc. Thus we find that *preparation for an examination* (especially in law), competition for an art prize and so on, very often end in neurasthenia. The *emotional* factor enters still more into the life of the speculator, who rushes from one excitement into another.

I have found that persons who are compelled to turn night into day are peculiarly apt to become neurasthenic, even although they have sufficient time to sleep during the day. This is the case with policemen, railway and telegraph workers, compositors, etc. Working in over-heated rooms is also a causal factor.

Any cause which lowers the general health may produce neurasthenia. Thus it often follows *loss of blood and lymph*, protracted febrile illnesses, and *infective diseases* of short duration (*e.g.* influenza). It is difficult to say to what extent this is due to the toxins. Diseases of the genital organs, the ear, the nose, and its accessory sinuses are frequently associated with neurasthenic symptoms. Most of the so-called reflex nasal neuroses are of this order. Persons suffering from chronic scoliosis are particularly apt to have neurasthenia, but in the experience of Oppenheim [1] and Petit, scoliosis is often a stigma of a neuropathic heredity. I have come to know a number of families many of whose members suffered both from scoliosis and neurasthenia ; most of these persons were highly, or even exceptionally intelligent.

There is no doubt that neurasthenia may have a *toxic* origin. Its symptoms may accompany chronic alcoholism, and it may be due also to chronic lead and arsenical poisoning and to excessive smoking.

Syphilitics often suffer from neurasthenia. The active causal agent in such cases is usually emotional excitement and nosophobic self-observation, but it is possible that the nervous system may be weakened by the infection itself and by the treatment. This causal connection is at least indicated by the fact that hereditary syphilis is often associated with neurasthenia (Binswanger). French writers (Charcot, Bouchard, Raymond) lay weight upon the gouty origin, and Raymond is particularly inclined to think that poisons originating in the organism are important causes of simple neurasthenia, which he distinguishes from psychasthenia (*q.v.*). Bouchard's view that the disease is produced by auto-intoxication in diseases of the gastro-intestinal system seems to me insufficiently founded, and still more does Glenard's [2] theory of its being due to changes in the position of the viscera (enteroptosis, gastroptosis (see below)).

[1] "Skoliose und Nervenleiden. D. Aerztezeitung," 1900.
[2] "Les Ptoses viscérales," etc., Paris, 1899.

Sexual excesses, and *masturbation* in particular, may prepare the way for neurasthenia; there is no proof that sexual abstinence has a harmful effect. This could only be the case in neurotic and highly excited individuals. The importance of congressus interruptus as a cause must be admitted.

Trauma is undoubtedly an important factor. Injuries to the head and concussion, especially if associated with mental excitement, as in a railway accident, frequently cause neurasthenia, but the traumatic form tends to be combined with symptoms of other neuroses. Neurasthenia also often develops after surgical operations.

Heat apoplexy may be followed by neurasthenia and other neuroses (Steinhausen).[1]

Symptomatology.—The chief symptom of neurasthenia is *irritable weakness*, *i.e.* abnormal excitability and fatigability. The exaggerated *excitability* is certainly general, but it by no means affects every part of the body in an equal degree. Unlike hysterical excitability, it is characterised by a certain constancy and uniformity. In the great majority of cases the feelings of discomfort, excitement, and tension are exaggerated, but the sensation of pleasure may also be increased. This exaggerated excitability is most in evidence as regards stimuli which affect the emotional life and the sensory spheres. Insignificant events, which make no impression upon a healthy mind, produce a condition of anger, irritation, anxiety, or depression. Ideas and recollections of various kinds may assume a distressing character on account of the accentuation of the feelings of aversion which accompany them.

Noises which, heard by a healthy person, do not affect his mood, produce unpleasant or even unbearable sensations. This may be exaggerated to such a degree that the sound of a human voice becomes insupportable and conversation is impossible. The irritability may, in the same way, be evident as regards optic stimuli, but the individual variability of the neurasthenic depends mainly upon the fact that in each case the troubles originate in and are specially associated with a different system.

Further, as Martius has emphasised, the increased excitability plays an important part as regards physiological stimuli arising from the internal organs, which under normal conditions are not perceived; the processes of digestion, of the action of the heart, etc., which in healthy persons produce no conscious sensation, may be felt to be uncomfortable and even painful. When this association has once been formed, the resulting disturbances will be intensified by the attention which is directed to the process. This has two results; on the one hand it enables the stimuli that come from these organs to pass more easily, and thus exaggerates the feeling of discomfort, and on the other this mental process has a harmful effect upon the automatic regulating functions which are mainly controlled by the sympathetic, and which not only do not require mental control, but are impaired in their action by it (heart, vascular system, etc.).

This abnormal excitability is associated with abnormal *fatigability*. This is the second great feature of neurasthenia, and it also more or less affects every part of the body. The unusually rapid exhaustion mainly affects the mental activities; the power of attention becomes quickly

[1] Leuthold's "Gedenkenschrift," ii., 1906.

exhausted and the capacity for perception is paralysed. The fatigability also involves the sensory functions and the muscular system. It is, however, less physical (as in myasthenia) than mental ; *i.e.* the feeling of discomfort produced by action comes on so rapidly and attains such a degree that activity is impaired or inhibited. The fatigability is thus to a certain extent a masked excitability.

In neurasthenia the influence of emotion upon the psycho-physical functions is also exaggerated, but these show only quantitative, not qualitative changes (no spasms, paralysis, etc.).

From this description it will be seen that neurasthenia is a *general disease*, but its symptoms and troubles may, to a certain extent, be *localised*. This depends mainly on two factors : 1. the site of the cause of the disease (*e.g.* sexual neurasthenia from excessive irritation of the genito-nervous system, etc.) ; 2. the congenital disposition, *i.e.* the irritable weakness may from birth—probably because of the inherited disposition —affect chiefly a certain part of the nervous system, *e.g.* in the abdominal region, which thus forms the starting-point of the disease. Possibly the congenital inferiority involves not only the nervous system, but also the muscles, glands, vessels, etc. That is to say, a certain organ may be so incompletely developed that from the first it is inferior and is particularly liable to be affected by the neurasthenia which develops sooner or later.

The *mental condition* of the neurasthenic has practically been described in the foregoing discussion, but the following details may be added :—

Mental depression is usually present, but is neither deep nor persistent. It is the result of the exaggeration of the sensations of discomfort and uneasiness and of the patient's reflections upon his morbid condition and the hypochondriacal thoughts thus produced. Periods of depression may therefore alternate with others of a normal frame of mind. The influence of one's *state of mind* upon one's physical and mental functions makes it easy to understand how uneasiness or depression may give rise to inhibitions and disturbances in various parts of the body, which in their turn produce other symptoms. This has been discussed at great length, *e.g.* by Goldscheider.[1] A *condition of anxiety* is very often present. The fear specially of a mental disease gives rise to an intense feeling of anxiety, which may even lead the patient to commit suicide. In many cases the anxiety only appears in relation to certain external conditions, *e.g.* in crossing a large, open space, etc. (consult the appendix as regards this and the accessory symptoms and causes of fear). The patient's irresolution and *incapacity for making a decision* are often so marked as to constitute a symptom of disease.

The *intellectual* powers are never greatly impaired, except in those cases in which the neurasthenia attacks congenitally weak-minded, psychopathic individuals. There are, it is true, frequent complaints of impairment of the power of perception, loss of memory, etc., but careful examination shows that the capacity for judging, reasoning, etc., is undiminished. The difficulty in directing attention to other things, in concentrating the thoughts, arises merely from the patient's constant preoccupation with his own condition. Thus he may read one passage two or three times without comprehending it, which explains why he apparently forgets things so easily. In addition to this the primary factor of exhaustibility already mentioned plays a part. The great majority of

[1] *Z. f. physik. Ther.*, x.

neurasthenics are incapable of doing sustained mental work; the feeling of fatigue comes on rapidly and prevents the work being continued. The intensity and duration of this fatigue is the outstanding feature of the disease. It may be exaggerated to such a degree that all mental work is absolutely impossible. I have often noticed this in severe cases of sexual neurasthenia and masturbation. Prolonged insomnia may have the same effect.

The power of bringing to mind visual memory pictures, of remembering the appearance of a certain person, a place, or an object, is sometimes notably impaired. Thus, an artist complained to me that he could no longer judge of a picture because he forgot one part whilst looking at another. Many of my patients have complained of this fragmentary apprehension of what they saw, and this condition sometimes produced a kind of compulsion to find the exact position of things. Poets and writers feel that they cannot develop their artistic imaginations, and that their power of conception and production is inhibited. In many cases this loss of perception is only felt at times, under the influence of excitement, etc.

Absence of the power of perception and loss or diminution of the sense of personality—"depersonalisation" (Janet, Österreich, *Journ. f. P.*, vii.)—may be one of the distressing mental symptoms in severe forms of neurasthenia.

In other cases the patient complains of the haste and instability of his thoughts—"flight of ideas." The wandering attention and the disconnected nature of his thoughts may be apparent in his conversation.

In neurasthenic children the capacity of sustained attention may be so impaired that, although very intelligent, they cannot learn, and are less advanced in their education than children of feeble mental powers.

One of my patients told me the remarkable fact that neither he nor the other members of his family could do two things at the same time, *e.g.* talk whilst dressing or undressing.

Further, we hear of *neurasthenic psychoses*. Thus Krafft-Ebing has described *transitory neurasthenic insanity*; Ganser, neurasthenic mental disorder, etc. Some of these conditions, however, are the psychoses of exhaustion, and others have not the features of mental disturbance, but represent forms of neurasthenia (Redlich). On the other hand hypochondria undoubtedly develops out of neurasthenia from which it can hardly be distinguished, and melancholia also arises out of neurasthenic depression. Consult Wollenberg, Nothnagel's "Handbuch," xii.; *C. f. N.*, 1905; and Friedmann, *M. f. P.*, xv.

The term *psychasthenia* (see later) is applied to the form of neurasthenia which chiefly affects the mental functions.

Headache, vertigo, and *sleeplessness* are the most prominent and common *subjective troubles*. There are very few cases in which these symptoms do not occur at some stage of the illness. The headache is usually described as a feeling of pressure on the head. The patient feels as if something ("a leaden weight, an iron plate") were pressing from within upon the brain or upon the skull, which feels as if it were splitting. In many cases there is merely a feeling of heaviness and dulness; the patient says, "My head is not free"; "I cannot think freely"; "There seems to be a mist over my thoughts," etc. They often complain that in talking they have a buzzing sensation in the head (not due, as in autophonia, to any disease of the tubes). The character of the headache may be greatly modified by *morbid introspection* and *nosophobia* (fear of illness). As neurasthenia is often associated with *hypochondria*, which often develops from the former, so the single symptoms of neurasthenia may become the source of hypochondriacal fancies. The headache suggests the presence of

a brain tumour or brain syphilis. The pain is greatly intensified if the attention is directed to it, and it may be felt by the patient just as he imagines the disease he is dreading would be felt. But the subjective character of the pain and its dependence upon the patient's attention and imagination can almost always be easily recognised. There are some cases in which the headache is brought on by any attempt at mental work; others in which it is connected with excitement, and in particular with unpleasant impressions.

Vertigo is a very common symptom. The patient usually complains merely of a momentary dazed feeling of losing his senses, of the floor giving way, etc. The giddiness is very often the result of fear. True attacks of vertigo with loss of balance occasionally occur; these are probably due to vasomotor influences. Ménière's disease is often related to neurasthenia; indeed, as I have found in many cases, there is a neurasthenic form of this condition (Politzer). Moreover, vertigo may come on when the position of the head is slightly changed, the patient lying on his side, his back, etc., a symptom which Ziehen rightly attributes to hyperæsthesia of the nerves of the labyrinth. The fear of vertigo may be so overwhelming that the patient will no longer walk out alone, and may never go out of the house again if his physician does not succeed in dispelling his fears by earnest advice and encouragement. In the case of a gentleman who had not left his house for a year on this account, my treatment by mental influence was so successful that within a short time he was taking long walks.

Insomnia is one of the most common complaints, and may on account of its persistence be the chief feature in the condition. The power of going to sleep is usually impaired. Sometimes the patient wakens too soon. In many cases the patient is not completely, but only half asleep. The degree of insomnia present is usually exaggerated by the patient, but chronic insomnia may be the result of neurasthenia. Many patients describe sudden starts of terror during sleep.

I have seen cases in which this symptom has been very distressing, when, for instance, the patient starts from sleep every quarter of an hour: in one of my cases the patient jumped so violently that he was often thrown out of bed. Another screamed so loudly immediately after falling asleep, that it became unbearable to the patient and those around him.

The *night-terrors* of childhood are often an early symptom of neurasthenia, but they may be due to other causes (worms, adenoids, etc.). Rey states that he has found adenoids in all his cases, but I think the symptom undoubtedly occurs chiefly in neurasthenic individuals, although it is not regarded as a definite symptom of neurasthenia.

Bad dreams may also be a sign of neurasthenia. One lady consulted me solely on this account, as for seven years her whole time of sleep had been disturbed by exciting dreams. Night and the time of sleep are indeed specially trying to nervous people, for many of the symptoms occur only at night or become exaggerated then. These include the condition of fear, nervous dyspepsia, urticaria, tinnitus aurium, pruritus, etc. This is due to various causes, one of which is that, in the absence of activity and of all external stimuli, the patient's mind is more than ever concentrated upon his physical processes. The importance of this factor has already been noted. Moreover, sleep is sometimes so superficial that it is disturbed even by the stimuli arising from the processes of digestion and peristalsis. It might be put thus, that in the same way as the susceptibility for alcohol, tobacco, and other poisons is increased, so is that for intestinal toxins, even the normal products of intestinal fermentation, etc., thus by their toxic influence giving rise to sleeplessness, pressure in the head, etc. Another possible factor is that under normal conditions the higher centres apparently exercise a regulating influence upon the subordinate (sympathetic) centres, which is diminished during sleep. Under pathological conditions the decrease in this regulating influence may be specially felt during sleep.

Some light has been thrown upon the condition of the circulatory system during sleep by plethysmographic investigations (Mosso, Brodmann, etc.). Brodmann (*Journ. f. P.*, i.) concluded from his experiments that the vasomotor activity is very independent in different parts of the body, and that this independence is greater during sleep than in the waking condition.

The accumulation of carbonic acid in the blood and the resulting impediment to the absorption of intestinal gas assumed by Lahmann (Stuttgart, 1905) does not agree with the facts established by physiology; but the theory contains a germ of truth, as the comparative slowness with which the blood circulates during sleep probably plays some part in the production of some of the neurasthenic troubles.

These statements do not, however, explain all the conditions. Thus I have treated a neurasthenic man who, for a number of years, had been wakened from sleep by a girdle-sensation, which occurred regularly at night while he was sleeping, never while he was awake. In another case a distressing pain came on behind the sternum, also only during sleep. I have observed a number of such cases. These *nyctalgias* or *hypnalgias*, as I have called them (*B. k. W.*, 1899), are exceedingly difficult to interpret.

It has already been mentioned that *narcolepsy* may be a symptom of neurasthenia. I have treated the head-master of a school who was obliged to prick himself during his teaching in order to keep from falling asleep.

Waking out of sleep may be associated with an unusual state of mind, a feeling of double consciousness, of confusion, somnolence, etc., of transient duration. Psychomotor inhibition, inability to speak, to move, etc., immediately after waking, may be signs of the neuropathic diathesis (Pfister, *B. k. W.*, 1903; see also Gudden, *A. f. P.*, Bd. xl.).

Disorders of the special senses may also show the exaggerated susceptibility and fatigability. The eye and ear are specially often affected. A dazed feeling, shimmering before the eyes, seeing spots ("mouches volantes"), or bright points, stars, and so on, rapid exhaustion in reading ("the letters swim together, dance before the eyes," etc.), and, more particularly, exaggerated sensitiveness to noises, buzzing in the ears, ringing, whistling, hissing before the ears, etc., are common, distressing and persistent troubles. Many of the children in one nervous family, descended from parents who were related, were troubled by always seeing a green wreath. Seeing and hearing may become quite painful. Other disorders, such as itching of the head, heaviness in the stomach, sweating salivation, a feeling of anxiety, etc., may occur when the eyes are fixed, especially in reading. Some of these "asthenopic disorders"—especially the fatigue caused by using the eyes—are apparently chiefly due to exaggerated fatigability of the muscles of accommodation and the internal recti. On the other hand the *power of seeing and hearing* is unimpaired, and ophthalmoscopic examination never reveals any disease of the optic nerves. I have only had one case of neurasthenia or hystero-neurasthenia under my own care in which the patient described a transient hemianopsia, and it is not impossible that the case might have been one of a combination with an atypical, abortive form of hemicrania. Slight narrowing of the field of vision has been found in a few cases of pure neurasthenia, and is as a rule a symptom of exhaustion—of the abnormally rapid fatigability. Special methods of testing (Förster, Wilbrand, Reuss) the condition of excentric vision will show the tendency to rapid exhaustion.

Nervous deafness may be associated with neurasthenia.

Hypersensitiveness to cold and especially to *heat*, *e.g.* to an overheated room, is a very common symptom. It may be so marked that a slight increase in the ordinary temperature of a room gives rise to discomfort. Hyperæsthesia to tactile and painful stimuli, *e.g.* the onychalgia

and trichalgia nervosa which I have described (*M. f. P.*, xiii.), may be a sign of the neuropathic diathesis and may accompany neurasthesia. On the other hand stimuli to which healthy persons are indifferent, *e.g.* stroking the hair, firm pressure, etc., may sometimes cause a pleasurable sensation.

As regards the *motor* functions, the usual symptoms are *weakness* (not paralysis), *tremor*, and *fatigue of rapid onset*. The patient usually complains of a feeling of weakness and of being easily tired. The power of voluntary movement is retained in all the groups of muscles. The strength as a whole is normal or but slightly diminished. Single movements are certainly carried out in a feeble, tremulous way, but the bearing of the patient shows that he is not exerting his whole strength; he either does not or cannot force himself to do so. The force with which he presses one's hand is often at first very slight, but if he is urged to increase it he can do so to a great extent. The reverse may occur; he may at first put forth a normal amount of strength, but the effort rapidly dies away.

Ziehen has demonstrated the exhaustion by the use of a dynamometer. He has found that it can be easily shown with regard to fixation and accommodation of the eyes, as when an object (pencil) upon which the patient has fixed his eyes is steadily brought nearer to him, double images begin to appear at steadily increased distances, and the point of near vision gradually moves farther away. We should also refer to the experimental observations of Mosso, Ballet, Philippe, Herz, Breukink (*M f. P.*, xv.), etc.

The motor weakness never develops into paralysis, and it is not limited to the muscles supplied by any particular nerve, but is practically always *general*. It is only confined to one side of the body (*i.e.* to the arm and leg of one side) or to the lower limbs under special conditions. It is not associated with atrophy or changes in the electrical excitability.

Tremor is very frequently present. It is generally of a *rapid, fine, vibratory* nature. In its slightest degree it may occur in healthy persons, especially after excessive smoking, drinking, or sexual indulgence. It accompanies active movements, but may also be brought on by mental excitement. A *fibrillary* tremor, affecting specially the orbicularis palpebrarum and orbicularis oris, the first interosseus, etc., is also common. It tends to appear specially in mental excitement or under the influence of cold, *e.g.* in the quadriceps femoris when the leg is exposed. It may develop into muscular agitation (myokymia [1]). Thus a patient who consulted me for neurasthenic melancholia had from childhood had myokymia of the calf muscles, which gave him no trouble. The attempt to stand with the eyes shut sometimes brings on tremor of the eyelids, which is hardly ever observed to the same degree in normal persons. The so-called essential hereditary tremor, which assumes various forms (Brasch [2]), is often related to neurasthenia.

The *tendon reflexes*, especially the knee-jerk, are usually exaggerated. Ankle clonus (as a rule slight and inconstant) may sometimes be elicited. There can be no doubt, however, of the fact, which I must emphasise in opposition to Babinski, Brissaud, etc. (see foregoing chapter), that a true ankle clonus may occur in rare cases of neurasthenia. The tendon

[1] There is apparently another form of myokymia of a rheumatic nature (Biancone), which I have several times seen in chronic muscular rheumatism. See also the section on muscular spasm.

[2] *Z. f. N.*, vii. See also the communications of Raymond (*Bull. méd.*, 1892); Raymond-Cestan (*R. n.*, 1901); Mitchell (*Journ. Nerv. and Ment. Dis.*, 1903); Schmalz (*M. m. W.*, 1905); Raymond-Thaon (*R. n.*, 1905); E. Neisser (*W. kl. R.*, 1906); Germ. Flatau (*A. f. P.*, Bd. xliv.).

reflexes are often markedly increased in the arms. On the other hand there are no other signs of muscular rigidity (spastic gait, the characteristic stiffness, Babinski's or Oppenheim's signs). It is only in great emaciation, such as may develop in exceptional cases from nervous dyspepsia, that the muscles may be very flaccid, causing the tendon reflexes to become so feeble that they can only be elicited with difficulty by the use of Jendrássik's method. *Absence of the knee-jerks always indicates the presence of an organic nervous disease* (see pp.1084 and 1094). It has been shown by Auerbach-Edinger and Oeconomakis, who examined cyclists after racing, that they may *temporarily* disappear after physical exhaustion.

Exaggeration of the mechanical excitability of the muscles and nerves is not uncommon.

There are no symptoms of paralysis of the cranial nerves. Speech as a rule is unaffected. In periods of mental exhaustion, and especially after sleepless nights, the patient may not be so eloquent as usual; he may occasionally have to search for a word, use a wrong one, or (both in speaking and writing) he may omit or displace a syllable or letter. But if his pronunciation of single words is tested, it will be found to be normal, and when the patient makes the effort, he can promptly repeat the most difficult catch sentence. Stuttering is often found in neurasthenia. There may also be a very transient form of mutism, which tends specially to occur in nervous children during school-hours, and is due to fear. Gutzmann has carefully described this form of lalophobia. Insufficiency of the internal recti is sometimes present. As a rule the *pupils* are normal, and the light reflex is always present and sometimes exaggerated. Slight *inequality of the pupils* may occur, or there may be marked spasmodic dilatation of one pupil (the reaction being always retained), but this is so unusual that it should always rouse suspicion of complication with an organic disease. A few cases (Pelizaeus, Riegel, Oppenheim) show it to be probable that the phenomenon of " springing pupil " (see p. 85) is in rare cases of a neurasthenic nature. Hippus is not uncommon.

Sensory Disturbances.—In addition to the headache described above, *pain* may be felt at various parts of the body. It is specially frequent in the back, and is then associated with tenderness of some or all of the vertebræ to pressure (spinal irritation), and erroneously referred to some disease of the spinal cord. It may occur in the extremities, the abdominal region, or the viscera. Neurasthenic pain is hardly ever very severe; it may be described as being violent, but any one who observes an attack, does not gain the impression that the patient is suffering intense pain. *Neuralgia* (sciatica, tic douloureux) may of course be combined with neurasthenia, and neuralgiform pain may develop at various parts of the body (see pp. 554 and 571). Jendrássik[1] describes neurasthenic neuralgia in terms similar to those which I[2] have used in describing the psychalgias. The pains described by Brissaud[3] as " habit-pains " also belong to this category. *Hemicrania* is very often associated with neurasthenia.

Paræsthesiæ, viz., tingling, formication, a feeling of cold (particularly in the feet), are seldom absent. They are often of a hypochondrical nature. The feeling of " going to sleep " may be very intense in the areas of certain nerves. These paræsthesiæ sometimes involve the ulnar region; they may be elicited by slight pressure in the area of any nerve,

[1] *D. m. W.*, 1902. [2] *B. k. W.*, 1898. [3] *R. n.*, 1903; *Progrès méd.*, 1904.

as the mechanical excitability of the sensory nerves is, in our experience, often increased in neurasthenia. The paræsthesiæ sometimes take the form of painful itching, which may lead to *pruritus* and *prurigo* of a very persistent type; these again may cause sleeplessness and so affect the general condition. *Pruritus ani* is a specially common symptom of neurasthenia, and is in my experience most frequently due to this cause. I have several times seen a combination of *prurigo*, *urticaria*, and *tic-like twitching of the muscles* in neurasthenics.

In one family the mother and three children suffered from pruritus ani et vulvæ; in each case it had developed in the thirtieth year, and none of the patients knew that the others were similarly affected. Another sister had a general alopecia.

There may be itching and tickling in the larynx which bring on attacks of coughing and thus prevent sleep.

Alopecia occurs so often in neurasthenia that there can be no doubt of its relation to this neurosis, but it may also develop from other causes.

The *vasomotor disorders* deserve special consideration. They are present in a number of cases. The patient complains of a feeling of *rush of blood* to the head or of troublesome *flushing*. This symptom is usually objectively evident. The face or one side of it suddenly (*e.g.* during the examination) becomes covered with diffuse spots of redness, which may spread over the neck and chest. The temperature of the skin is at the same time raised. The patient is usually conscious of this disorder, and the mere thought of flushing is sufficient to bring it on. This *fear of flushing* (Bechterew[1]), erythrophobia, or *phobie de la rougeur* (Pitres et Regis) may be a very distressing and persistent symptom. Vasomotor disorders play a specially important part in *neurasthenic conditions of anxiety* (compare following chapter). It is a common thing for slight cutaneous irritation to cause severe and persistent redness of the skin, and sometimes the formation of weals (*urticaria factitia*, *dermographia*). It should be mentioned that there is a form of *spontaneous* urticaria of neurasthenic origin, which is very common in nervous children. The urticaria which is brought on by eating certain foods (strawberries, eggs) may also be due to the neuropathic diathesis.

A hæmorrhagic form of urticaria factitia combined with other severe symptoms of the vasomotor nervous system was seen in one case. Spontaneous appearance of ecchymoses may occur in rare cases of neurasthenia. Fürstner (*Mitt. a. d. Grenzgeb.*, xi.) has described interesting forms of vasomotor disorders of a hereditary, family character.

The rush of blood to the head may be associated with pulsation and buzzing which is felt all over the head or at certain places.

It has already been mentioned on p. 586 that there is a neurasthenic form of intermittent claudication. I have recently seen one such case, in which the symptom repeatedly disappeared when the patient was staying in the mountains.

The disorders of the *nervous system of the heart* are closely related to the vasomotor disturbances. These are both subjective and objective. Chief amongst the *subjective* troubles is palpitation of the heart. The patient feels as if the action of his heart were accelerated and excited. This sensation is usually, though not always, accompanied by acceleration of the heart's action. Less frequently he feels as if the heart stopped for a

[1] *Oboszrenje*, 1895; *N. C.*, 1897.

moment, and this may be associated with an actual interruption of the heart beat. If these attacks are prolonged, precordial anxiety, want of air, pains in the region of the heart and radiating into the arm, paræsthesiæ, and a feeling of cold in the hands and feet, etc., may also occur, and the condition may greatly resemble angina pectoris. Nervous angina pectoris has been thought to be due to angiospastic conditions in the coronary arteries. Severe attacks of this kind, with angiospasm of the peripheral arteries, and transitory dilatation of the heart (even with transient swelling of the thyroid during the attack) have been described by J. Jacob. According to Curschmann [1] similar symptoms may occur in true angina pectoris.

Acceleration of the action of the heart may often be recognised *objectively*. Mental excitements in particular, and also errors in diet, the use of coffee, slight physical exhaustion, etc., may markedly increase the frequency of the pulse. I have often had under my care for nervous palpitation persons who gave way to sexual excitements accompanied by fear and remorse. In addition to masturbators, married people were specially affected whilst attempting illicit sexual intercourse by palpitation of so violent a nature that they were compelled to abstinence or to conjugal fidelity. *Attacks of tachycardia* may occur apparently quite independently of mental influences. They may come on without warning, in the middle of some indifferent conversation. The pulse becomes suddenly *small, weak*, and *very frequent*; it may reach 200 beats in a minute, as I have seen in one very severe case, in which it was very small and at times hardly perceptible. The symptom was in itself very alarming, but the patient was able to go on conversing quietly, breathing comparatively easily and taking his food. Although the pulse beat remained at this rate for five to six hours, the patient's general condition was practically unaffected. He only felt slightly giddy during a rapid change of position, but could walk a considerable distance. I was certain that morbid introspection was not a factor in this case, as the patient was in comparatively good spirits and conversed of matters which necessarily claimed his whole attention.

As regards the nature and cause of so-called *paroxysmal tachycardia*, many different opinions have been expressed (Bouveret, Nothnagel, Hoffmann, Hochhaus, Martius, Rose, Merklen, Maixner,[2] H. Schlesinger,[3] Singer,[4] A. Hoffmann,[5] and others). Attacks of rapid palpitation, sudden in their onset and cessation, are accompanied by marked acceleration of the pulse beat, which may reach 200 to 260 per minute, and may show a peculiar regular "embryonal" rhythm, like that of a pendulum. The heart limits are usually normal, but they may be distended. These attacks last for hours, but occasionally persist for many days. Schlesinger speaks of a status tachycardicus. Transient glycosuria and albuminuria have been observed by Kraus and Huchard after the attacks. The course is usually favourable, but the attack may endanger life (especially if heart-disease is present). Goldscheider [6] and Schlesinger therefore regard the prognosis as grave. I know one case in which these attacks have persisted for twenty years without leading to any further trouble. It is doubtful whether all the cases described by this name actually belong to this class. Most of the cases point to the nervous origin of the attacks, and the affection is therefore regarded by most writers as a neurosis (vagus-neurosis, bulbar neurosis). Nothnagel compares it to epilepsy. Schlesinger points out that it may occur

[1] *D. m. W.*, 1906.

[2] Sbornik Klinický, iii.

[3] Volkmann's *Samml. klin. Vortr.*, No. 433, 1906 (including also his earlier work and the literature).

[4] *W. kl. R.* and *W. m. W.*, 1903.

[5] *Z. f. k. M.*, Bd. liii.

[6] "Die Diagnose des gesunden Herzens." Leuthold's Gedenkschrift, i. See also *Z. f. phys. u. diät. Therap.*, 1906.

in combination with epilepsy, exophthalmic goitre, and Quincke's œdema, and assumes that it may represent an equivalent of an epileptic attack. In one case he found pathological changes in the vagus and nucleus ambiguus. Martius regards the stretching of the muscles of the heart as the primary element, but this is present only in one group of cases. Others would distinguish two types of the disease, viz. a cardiac and a nervous, etc. In any case similar conditions occur in heart-disease. The attacks may be brought on by dyspepsia and by excesses. In a case of Schlesinger's, they could be cut short by pressure on the vagus and in another by forced inspiration.

The influence of *respiration* upon the pulse is often notably increased in nervous persons; this applies specially to lengthening of expiration (Thomayer, Vaníysek [1]). Stimuli affecting the trigeminus or the mucous membrane of the nose, *e.g.* an inhalation of ammonia, may, as Kraus has shown (*D. m. W.*, 1905), considerably increase the rate of the pulse and the blood-pressure in neurasthenics.

Palpitation may occur during sleep or just as the patient is falling asleep, and thus produce insomnia. Bouveret speaks of a permanent neurasthenic tachycardia, which, however, I regard as a very rare occurrence.

The pulse sometimes becomes slow or intermittent, and then accelerated as the patient bends down; less often it is at first accelerated (Oppenheim,[2] Erben [3]). Erben found the pulse in such a case slow and abnormally tense, and he therefore speaks of a characteristic vagus pulse, which he ascribes to venous hyperæmia of the brain (?).

Inequality of the contractions (allorhythmia) is sometimes present, and *arhythmia*, irregular action of the heart, and much less frequently *bradycardia*, have been noted in neurasthenics and hysterics, especially as complications of dyspepsia. In most of the cases which I have treated for cardiac neurasthenia, the arhythmia has been of short duration and usually attributable to mental causes, in particular morbid, anxious introspection. But, it may be brought on by other causes, such as gastric disorders, meteorism, physical exhaustion. Irregularity of the action of the heart and an intermittent pulse are often due to nicotin poisoning. In a case of *bradycardia* described by Grand, the pulse rate fell during the attacks to 27 per minute. Ziehen reports chronic bradycardia, in which the pulse rate was 43. In my experience this symptom has been rare and usually of transient occurrence, but a habitually slow pulse may be found in healthy persons. I would specially refer to Neusser's [4] careful study of this question. Bradycardia may be associated with a sensation of fear and oppression, and notably with vertigo. The so-called Stokes-Adam's symptom should not be discussed here (see p. 675).

Heart murmurs of neurasthenic origin are very rare, but at the height of some excitement an extremely rapid heart may be associated with a systolic murmur. This, however, is quite inconstant. According to Drummond, these murmurs are chiefly of a cardio-pulmonary nature, *i.e.* they only accompany respiration. Marked pulsation of the vessels may also occur in neurasthenia. Indeed, strong pulsation of the abdominal aorta has led to the diagnosis of aneurism and to surgical operation (Hösslin). The pulsation sometimes extends more or less over the whole body and is distressingly intense (Dana). Pulsating shaking of the head (or Musset's symptom) and so-called pseudo-aortic insufficiency may

[1] *Arch. bohém.*, 1903; *W. kl. R.*, 1905.

[2] "Die traumat. Neurosen," first edition, 1888.

[3] *W. kl. W.*, 1898; see also Vaníysek, *Arch. bohém.*, vi.

[4] "Ausgewählte Kapitel der klinischen Sympt.," etc., I. Bradykardie, etc., Wien, 1904. See also Mackenzie, "Study of the Pulse," 1902.

also occur in vasomotor neurasthenia. The nervous disorders of the heart are so prominent in some cases that they have been termed *neurasthenia cordis*. In this respect Gerhardt's statement, that more than half the patients who have consulted him on account of palpitation, suffered, not from valvular defects, but from some functional disease of the heart, is an interesting one. Abnormal mobility of the heart may be a sign of neurasthenia and a cause of the symptoms just described (Determann, Hoffmann, Rumpf).

Statements as to acute dilatation of the heart under the influence of mental excitement (Stark, *M. m. W.*, 1905; Kress, *N. C.*, 1905) should be accepted with great reserve. Goldscheider is also sceptical with regard to them.

An attempt has been made to refer the cardiac and vascular symptoms—the *neurasthenia cordis* and *vasomotoria*—to functional derangements of the vagus and sympathetic nerves and to regard them as a special class (Gerhardt, Riegel, Lehr, Determann, etc.). Lehr [1] has gone particularly far in this direction, but it is as yet hardly possible to analyse the symptoms of the various attacks in this way and to distinguish between a stage of irritation and one of paralysis.

Recent writers mostly adopt Engelmann's theory, which is that the action of the heart is regulated by influences of four kinds: 1. chronotropic, which control the stimuli and therefore the rate of the heart beat; 2. bathmotropic, which affect the stimulation threshold of the stimulus which excites contraction; 3. dromotropic, which modify the power of the heart muscle to convey stimuli; and 4. imotropic, which influence the power of contraction itself. On this subject see F. Kraus, *D. m. W.*, 1905; Volhard, *D. m. W.*, 1905.

Fuchs found that a simple muscular action, such as that produced by raising the extended leg, produced arhythmia, shown by the sphymograph, which he attributed to instability of the vagus centre; he found also that an allorhythmia, which disappeared when the patient lay on his back, was immediately produced by movements of resistance.

The data regarding the condition of the blood pressure are conflicting (Strauss, Haskovec, Rumpf, Kraus, Federn). All that can be said is that in cardiac and vasomotor neurasthenia the blood-pressure is very variable, is excessively influenced by mental excitement and change of position (Oppenheim, Bing,[2] Kraus, etc.), and that is not infrequently very marked in degree. In some cases of vasomotor neurasthenia I have registered a pressure of 160 to 200 with Gärtner's apparatus. It is much less often abnormally low, but I do not think that Fleury has succeeded in his attempt to distinguish two different forms of neurasthenia according to the condition of the blood pressure.

Tachypnœa may also be a symptom of neurasthenia (André, Reckzeh, Hofbauer, Pal [3]).

There can be no doubt of the relation of *asthma* to neurasthenia or to the neuropathic diathesis. The term has, it is true, been applied to various conditions, such as cardiac asthma, which do not belong to this group, but the typical form of bronchial asthma is now generally regarded as a neurosis. This is indicated by the general condition of the asthmatic patient, the psychogenic origin of the attacks, which can at least be ascertained in many cases, and their alternation with other nervous conditions, such as hemicrania, urticaria, agoraphobia, etc. The majority of persons suffering from asthma are neurasthenics, and it may indeed be one of the earliest manifestations of the neuropathic diathesis. There are, of course, many indications that there is an underlying special disposition—a special instability of the bulbar respiratory centre (and the adjacent vaso-motor centres). This is congenital or acquired from certain causes. Irritations coming from the respiratory tract itself

[1] "Die nervöse Herzschwäche und ihre Behandlung," Wiesbaden, 1891.
[2] *B. k. W.*, 1906.
[3] *W. m. W.*, 1906.

may produce this condition in the respiratory centre. These include chronic bronchitis and certain affections of the nasal mucous membrane (polypi, swelling of the mucous membrane beside the turbinal, possibly changes in the nerves of the nasal mucous membrane). Asthma dyspepticum, uterinum, etc., have also been described. The attacks come on *spontaneously* (*i.e.* without any recognisable external cause), particularly during the night, or as the result of some *exciting cause*, *e.g.* substances which irritate the nasal mucous membrane and the olfactory nerve (smelling certain gases, the pollen of plants, as Trousseau personally found, etc.), acute catarrh, and above all mental factors. Although many asthmatics only suffer from attacks when in certain places and are quite free from them in others, there is no doubt that climate plays merely a subordinate part in their causation. The idea or the fear of an attack which is associated with certain places is a much more active agent. The influence of emotion upon the respiratory centres is a physiological one; in this case it is exaggerated and modified in such a way that emotion of a certain kind brings on an attack of asthma. The idea awakens the state of feeling which finds its expression (in Kohnstamm's sense) in the asthmatic attack. There is a great resemblance therefore between the conditions of asthma and agoraphobia. Its occurrence only on certain days (*e.g.* only on Mondays) also points to its psychogenesis. One of my patients only suffered from asthma in certain houses.

The attack comes on suddenly, or is preceded by a feeling of oppression, pressure in the head, etc. The chief feature is dyspnœa, which is mainly expiratory. The breathing is not as a rule accelerated, but is deep and slow, the latter especially in expiration. The want of air is shown by marked contraction of the accessory muscles, redness or cyanosis of the face, an anxious expression, and often by hyperidrosis and secretion of tears. The pulse is usually small and frequent. The patient cannot lie down; he sits up in bed or at the window, and is entirely under the control of his need for air. On auscultating we find sibilant, sonorous, and other rhonchi. Percussion shows the diaphragm and the liver to be low, and the margins of the lungs to be enlarged (from abnormal inflation). Examination of the blood reveals increase of the eosinophile cells (F. Müller [1]). There is usually some expectoration in the course of, and particularly towards the end of the attack. The secretion is scanty, viscous, and mucoid, and often contains so-called asthma spirals and Leyden's crystals. As the expectoration increases, the attack usually diminishes, ending on an average in several hours, when the patient's condition becomes quite normal.

But the attacks may last a very short or a very long time, and there are various varieties and abortive forms (*e.g.* simple snuffing with a marked impulse to sneeze). Loss of consciousness and epileptiform convulsions at the height of the attack only occur in rare cases.

The prevailing view is that asthma is due to a spasm of the muscles of the bronchi or the diaphragm, or possibly of both. But the vasomotor element is also present. A. Fränkel regards the hyperæmia and swelling of the mucous membrane of the fine bronchi and the secretion as the chief features. The importance of vasomotor disorders is also emphasised by Brissaud and Schestopal. Einthoven attributed acute inflation of the lungs to spasm of the bronchial muscles and to irritation of the vagus, and Sihle found diminution of the blood pressure. He

[1] *Z. f. ärztl. Fortbild.*, 1906.

thinks asthma is a neurosis, made up of four main elements: 1. hypertonia of the smooth muscles of the respiratory tract; 2. hypertonia of the muscles of the vessels and heart; 3. a bronchial secretion, and 4. hyperæmia of the bronchial mucous membrane. Sihle (*W. kl. W.*, 1903) has also fully considered the psychogenic origin. Among the recent contributions to the subject of asthma we may mention those of A. Schmidt, "Bronchialasthma," Würzburg, 1903; Barbarin, *Thèse de Paris*, 1904; Dunbar, *B. k. W.*, 1904; Brügelmann, *Therap. Monatsch.*, 1906; Strübing, *D. m. W.*, 1906; Siegel, *Therap. Mon.*, 1906; A. Wolff, "Das Heufieber," etc., Senator-Festschrift, and *D. m. W.*, 1906; Goldscheider, "Vortrag in Fortbildungskurs," 1907; Strümpell, *Med. Klinik*, 1908. See also Kuttner, "Nasale Reflexneurosen," Berlin, 1904.

The *sexual system* is very often the starting-point of the nervous troubles. The mode of origin is usually as follows: *masturbation* is the first link in the chain, or is in itself a symptom of the neuropathic disposition, especially when the impulse has been awakened in early childhood.[1] Masturbation gives rise to *seminal emissions*. If these occur frequently, perhaps every night or three to four times in the week, and if the semen is passed several times in one night, the nervous system may be directly affected and neurasthenia produced. *Hypochondria*, however, usually plays an intervening part. The patient becomes alarmed by the pollutions, *reproaches* himself for the habit he has acquired, is afraid that he has or will have some disease of the spinal cord, and becomes morbidly attentive to his symptoms, and thus the neurasthenia is produced.

It should be remembered that the higher degrees of onanism, and especially those beginning in early childhood, occur in persons who are congenitally nervous or even degenerate (imbecile, psychopathically defective, or showing signs of the insanity of degenerates). Personally, I have no doubt that the tendency to masturbation may itself be inherited. Thus, in many cases we have a *vicious circle*: the tendency to masturbation is in itself a symptom of the neuropathic diathesis, and the masturbation again gives rise to a crowd of nervous troubles.

Spermatorrhœa, the loss of semen during the day after urination, and specially after defecation, the ejaculation being unaccompanied by erection and orgasm, should be distinguished from seminal emissions, although the two are often combined. It has been thought that such cases were usually due to prostatorrhœa, but careful investigations have shown that this secretion often contains spermatozoa. It is not improbable that spermatorrhœa is due to paresis of the ejaculatory duct. Actual ejaculations rarely occur during the day apart from defecation, but on the other hand it is not unusual for small quantities of semen to pass in drops during physical exertion. Some of my patients stated the remarkable fact, that as school children they had erections and spermatorrhœa when doing difficult mathematical problems. I have found numerous forms of these perversities of psycho-physical functions in degenerative neurasthenia. In one neurasthenic man an emission unaccompanied by sexual feelings had occurred for about eight years every time he had his hair cut.

[1] The tendency may even appear in infants. Indeed I have treated a child whose mother had noticed stiffening of the penis with congestion of the face, sweating, and other signs of excitement, in the second month. The child had a very marked heredity, the grandfather and great-grandfather being alcoholics, an aunt insane, an uncle showing pathological alcoholic conditions, the mother, aunt, and grandfather having abnormally small hands with very short, broad, terminal phalanges. When the child was brought to me at the age of six to seven, he suffered from conditions of fear, fixed ideas, and pathological habits. The tendency to masturbation still persisted. Fürstner is inclined to regard vasomotor disturbances in the genital apparatus as the primary element, and to hold them responsible for the premature masturbation.

Continuous spermatorrhœa is only observed in severe traumatic affections of the spinal cord, and then only in very rare cases.

Spermatorrhœa may also be the result of onanism, but *chronic gonorrhœa* with inflammation of the prostatic part of the urethra is very often present; this in itself may cause spermatorrhœa, very specially in masturbators or neurasthenics. The circle widens when we remember that emissions and spermatorrhœa may not only result in neurasthenia, but may be increased and kept up by it. *Thus the circuit of sexual neurasthenia becomes complete.*

Fürbringer (Nothnagel's "Handbuch," xix.) speaks of the onanistic neurosis. Krafft-Ebing have made a laboured attempt to divide the development of sexual neurasthenia into three stages: (1) that of a local genital neurosis, (2) that of a neurosis of the lumbar cord, and (3) that of a general cerebro-spinal neurasthenia. Peyer, Hammond, Eulenburg, Gyurkovechky, Loewenfeld, and others have carefully studied this question. I must not omit to mention that I have known some patients in whom masturbation, acquired in early childhood and persisted in until the thirties, had resulted only in slight neurasthenic symptoms.

The chief nervous troubles are *headache*, *backache*, *depression*, *disinclination for any activity*, *absent-mindedness*, *shyness*, and a *feeling of physical weakness* and *mental vacuity*. To these are added various other symptoms which give rise to the fear of spinal disease; this dread is the characteristic feature of the condition. Neurasthenics of this class often complain that coitus is followed by pain in the back which lasts for a whole day. Nervous cardiac symptoms, vasomotor disorders, and nervous dyspepsia are common symptoms of sexual neurasthenia. *Erections* occurring specially during sleep may be a distressing symptom. In some of my cases they were the chief cause of the sleeplessness. A few observations show (Mainzer,[1] etc.) that they may persist for days and weeks and be associated with great discomfort.

Impotence is a frequent result—and also a cause—of sexual neurasthenia. Indeed, it is not too much to say that the majority of persons complaining of impotence are neurasthenics, and that they usually suffer from abnormal seminal losses. The impotence is usually only relative and temporary. The desire as well as the power of erection may be diminished, or the ejaculation takes place too quickly, possibly before insertion, less frequently it is too long delayed. The mental element is very often, perhaps in most cases, the main factor. The idea of failure arises before or during the act, and this has an inhibiting effect and disturbs the course of the physiological processes which accompany coitus. Impotence may also be the result of sexual excesses, in particular of *unnatural sexual intercourse* and perverted sexual feelings, and may be either absolute (paralytic impotence) or relative, *i.e.* natural sexual intercourse may be impossible, whilst the sexual impulse can be satisfied in unnatural ways. The various perversities, peredasty, sadism, exhibitionism, flagellantism, etc., which are usually due to mental degeneration, will not be discussed here.

Although impotence may not be present, the power of procreation may be prevented by absence of spermatozoa. According to Fürbringer, azoopsermia is a common result of prostatorrhœa, as it is the prostatic secretion which preserves the vitality of the spermatozoa.

Although this description of sexual neurasthenia refers chiefly to men, there is no doubt

[1] *D. m. W.*, 1903. With regard to priapism and its various causes, see also Goebel, *Mitt. aus Grenzgeb.*, xiii.

that excessive masturbation is common in girls and women, and that, as in man, it may give rise to neurasthenia and hypochondria. But secretions from the glands of Bartholini (Krafft-Ebing,[1] Bernhardt[2]), resembling pollutions, are by no means so regular an occurrence, nor have they any comparable influence upon the general health. One woman, who showed other symptoms of hysteria and neurasthenia, consulted me because she became unconscious during coitus.

On this subject consult Forel, "Die sexuelle Frage."

The *functions of the bladder* are not markedly affected in neurasthenia. There is occasionally abnormal irritability of the bladder,[3] and even slight quantities of urine cause strangury and even dysuria. Here also the idea and abnormal introspection are the cause of the troubles. Thus the fear that the impulse to micturate will occur at an inconvenient time, *e.g.* in the theatre, will bring it on in a marked way (compare the chapter on conditions of anxiety and imperative ideas). One of my patients was obliged to micturate whenever he heard the sound of the water running from a tap. (Another, who suffered from bladder weakness of an organic nature, could pass urine without trouble under the same conditions.) A neurasthenic physician, who was afraid of diabetes mellitus, was obliged to urinate about twenty times a day, although there was no increase in the quantity of urine passed. In addition to this *pollakuria*, persons suffering from neurasthenia may be unable to pass a drop of urine under certain circumstances, particularly in the presence of others (*Harn-stottern, bégaiement urinaire* of Paget).

It may also be mentioned that the *enuresis nocturna*[4] of childhood has often a neuropathic cause. It may be inherited and familial (Guyon). I have treated a youth of seventeen for this trouble, whose twin-brother suffered in the same way. Many writers (Guyon, Frankl-Hochwart-Zuckerkandl) think it is due to congenital weakness of the sphincter vesicæ. Others (Trousseau, etc.) attribute it to hyper-excitability of the detrusor vesicæ or a lack of proportion in the innervation of the detrusor and of the sphincter vesicæ (Bokai, Ultzmann, etc.). Dittl regards incomplete development of the prostate and the sphincter internus and a large secretion of urine due to a fluid diet as the cause. Kuttner has traced the trouble in some cases to a chronic reflex spasm of the sphincter externus. Other factors are the uric acid diathesis, phosphaturia, vulvitis, adenoids, etc. I regard the nervous disposition as the essential cause and think it is specially due to hyper-excitability of the sympathetic nervous system or to defective regulation of the sympathetic bladder centres by the spinal and cerebral centres. Pfister (*M. f. P.*, xv.) thinks enuresis nocturna is a stigma of the neuropathic diathesis.

Enuresis nocturna is not infrequently associated in nervous children with a diurnal, and specially with an emotional enuresis (micturition on excitement, etc.). In nocturnal enuresis the urine is not passed in drops, but as a rule in large quantities.

Disturbances of the *digestive functions* are specially outstanding phenomena of neurasthenia. *Nervous dyspepsia* (Leube[5]) is not, indeed, an independent disease, but is one of the most common symptoms of

[1] *W. med. Pr.*, 1888.
[2] *Arztl. Praxis*, 1904.
[3] Literature in the review by Hirsch, *C. f. Gr.*, 1904.
[4] See the bibliography in the review by Landau, *C. f. Gr.*, 1903; and Frankl-Hochwart-Zuckerkandl in Nothnagel's "Handbuch," xix.
[5] *A. f. kl. M.*, 1878, and *B. k. W.*, 1884; Leube-Ewald, "Verhandl. d. iii. Kongr. f. inn. Med.," 1884. For literature see Riegel, Nothnagel's "Handbuch," xvi. 2, p. 852.

neurasthenia. At first and in slight cases the troubles, which do not occur regularly, but come on sometimes after meals, consist in a feeling of repletion, or an uncomfortable sensation of pressure in the region of the stomach, sometimes accompanied by eructations, a sour taste, etc. The appetite may be normal or absent, and is usually very capricious. Distressing thirst may be present. In some cases there is pain or hunger, and discomfort if the stomach is empty (gastralgokenosis of Boas). There may be bulimia. The disorders are often brought on by slight errors in diet; in other cases they may be absent in spite of prolonged debauches. Mental excitement (anger, worry, etc.) is not only a frequent cause of the condition (emotional dyspepsia of Rosenbach [1]) but often the exciting cause of the various attacks of indigestion and gastric disorder. Pawlow, with whom Bickel [2] and other agree, has thrown much light upon these relations. The general condition and strength are generally unaffected, except in severe cases. In these all the disorders already named are intensified and characterised by their persistency. Troublesome eructations, vomiting, cardiac discomfort and meteorism may develop. Less and less food is taken, partly because the patient is afraid of the results of eating, partly because his appetite diminishes, until eventually complete *anorexia* develops and leads to emaciation and loss of strength.

Investigations into the *chemistry of the stomach* and its motor functions in these conditions have not led to any definite results. There is undoubtedly a condition of *hyperacidity* of the gastric juice. The secretion of hydrochloric acid may undergo paroxysmal increase at long intervals and lead to attacks of severe pain, ending in the vomiting of sour matter (Rossbach's gastroxynsis). Want of acidity has also been observed.

Strümpell does not attach great importance to these symptoms, which may occur in normal conditions. There is great diversity of opinion and experience as regards the continuous flow of gastric juice (*gastrosukkorrhœa* or Reichmann's disease) (Reichmann,[3] Stiller, Ewald, Bouveret, Schreiber, Riegel, Boas, Martius, Sahli, Strauss, Albu, Pickhardt, L. Küttner,[4] Krauss,[5] and others), but hypersecretion of the gastric juice, associated with persistent, exacerbating symptoms, such as pain in the stomach, vomiting, thirst, constipation, etc., apparently occurs independently of eating. See also Boas, "Bemerkungen über den digestiven Magensaftfluss" (*D. m. W.*, 1907), and Graul, *Würzburger Abhandl.*, 1905.

The process of digestion may be retarded, although the gastric secretion remains normal. Rumination or merycism may also be a symptom of neurasthenia. It was a transient disorder in some of my cases, and has been observed in a familial form (Müller).

Valuable contributions to this subject have been made by Boas, Jürgensen, Einhorn, Levi, and Lederer.[6] As regards the relation of hæmophilia to rumination, see the comprehensive work of Presslich [7] and the paper by Brockbank.[8]

Haste in eating and inability to masticate slowly, etc., may be signs of the neuropathic disposition. I have seen some cases characterised by troublesome bulimia.

Constipation is usually present, and it not infrequently precedes the onset of the neurasthenia for years and decades. It may possibly be one of its causes. On the other hand, this trouble is often inherited and

[1] *B. k. W.*, 1897. [2] *D. m. W.*, 1905; *B. k. W.*, 1905. [3] *B. k. W.*, 1882 and 1884.
[4] *B. k. W.*, 1905. [5] *D. m. W.*, 1907. [6] *W. kl. W.*, 1904.
[7] *W. m. W.*, 1904. [8] Abstract, *D. m. W.*, 1907.

familial. Sluggish peristalsis (Kussmaul[1]), atony, or less often spasm (Cherchewski, Westphalen) of the intestinal muscles are usually the cause. *Flatulence*, the feeling of flatulent distension of the bowels with a tendency to eructation and a desire to put up flatus, is a very frequent complaint. These troubles often come on in the late hours of the evening and prevent the patient sleeping. A part is played in addition to the exaggerated sensibility and anxious self-observation, by the atony of the intestine and the distension caused by the accumulation of gas, which again prevents absorption (Nothnagel, A. Schmidt). Strümpell, however, attributes all the symptoms of nervous dyspepsia to mental influences. A tendency to diarrhœa is rare, but there are some nervous persons who suffer from attacks of it on any excitement (especially suspense) or immediately after taking any food. Mucous masses, membranous tubes, and shreds may be found in the fæces.

An attempt has been made to distinguish between mucous colitis and membranous enteritis (Nothnagel, Rosenheim, Ewald, Boas, Fleischer, etc.), and to regard the former only as a pure neurosis, the latter being ascribed to an intestinal catarrh. This distinction cannot be fully carried out (Leube, Westphalen). Ewald speaks of myxoneurosis of the intestine (*Ther. d. Geg.*, 1907). See also Boas (*D. m. W.*, 1905), Albu (*Ther. d. Geg.*, 1906). According to Albu (*B. k. W.*, 1905) achylia gastrica may be a stigma of degeneration.

Neuralgic pain in the abdominal organs is not often complained of. Pressure points, which seem to correspond to the sympathetic abdominal ganglia, are often found.

Fürbringer mentions a nervous hepatic colic, paroxysms of which may resemble gall-stones. Such cases have also been described by Pariser, Fuchs, Forster, Ewald,[2] etc., but some of these are really hysterical. The diagnosis is made chiefly from the other neurasthenic symptoms (*e.g.* absence of jaundice, swelling of the liver, etc.). The cases attributed to neuroses of the cœliac plexus (F. A. Hoffmann, Buch, Mackenzie,[3] etc.) require further explanation. An attempt has even been made to attribute cholelithiasis to a nervous cause (Glaser), and I have been surprised to find how often this trouble occurs in nervous women and how little effect surgical treatment has upon the neurasthenia.

We must admit that protracted affections of the gastro-intestinal tract may give rise to symptoms of neurasthenia, as Herzog in particular has shown, but this mode of onset is on the whole very insignificant as compared with the frequency of nervous dyspepsia.

The relations of enteroptosis and gastroptosis to nervous dyspepsia are also obscure. Glénard's[4] view that they cause neurasthenia is untenable. Changes in the position of the gastro-intestinal tract and the viscera, floating kidney in particular, may probably cause symptoms resembling those of neurasthenia, as Ewald, Bouveret, Boas, Einhorn, Aufrecht, Smith (*Brit. Med. Journ.*, 1906), and others have reported. General emaciation, flaccidity of the abdominal walls and ligaments, and even dilatation of the stomach may result from nervous dyspepsia, anorexia, and atony, and gastroptosis may develop from this cause. It has been suggested that both conditions are congenital and develop independently of each other out of the neuropathic diathesis (Stiller, Rosengart). Abnormal mobility of the tenth rib (floating rib) is said to be a frequent accompaniment of these conditions, but this has been contradicted (Meinert, etc.). In many cases there is no connection between these abnormalities and neurasthenia, and they may undoubtedly exist without any sign of nervous dyspepsia being present (Strümpell).

[1] Volkmann, *Samml. kl. Vortr.*, 1880, No. 181.
[2] "Mod. ärztl. Bibl." 1904; and *Therap. der Geg.*, 1906.
[3] *Brit. Med. Journ.*, 1906.
[4] "Les Ptoses viscérales," etc., Paris, 1899.

It is not certain whether spasm of the œsophagus, mentioned in the section on hysteria, may also be caused by neurasthenia. Severe cases of this kind have been observed, some even ending fatally from inanition, in which there was no organic change in the œsophagus or heart. As regards cardiac spasm, see the comprehensive paper by Mikulicz (*D. m. W.*, 1904) and H. Strauss (*B. k. W.*, 1904).

Hyperidrosis, either general or limited to certain areas of skin, is a frequent symptom. It may be very marked and persistent. A case of F. Müller's,[1] in which general hyperidrosis led to exhaustion and death, is quite unique. *Salivation* is an occasional, and abnormal dryness of the mouth a more common complaint. Salivation may, when very severe, be a very troublesome symptom. Neurasthenics often complain of a constant or transient feeling of moisture round the mouth, anus, etc. From personal experience, I have no doubt of the existence of a nervous form of cold with attacks of sneezing and much secretion. I have seen this alternate with other vasomotor disturbances. *Polyuria* and *polydipsia* may also develop during neurasthenia, and transient *glycosuria* may be one of its symptoms. We have already referred to the occurrence of alimentary glycosuria. Albuminuria due to mental excitement has been noted in rare cases, but it should not be forgotten that slight albuminuria may occur under physiological conditions (Leube). Oxaluria has also been described; large quantities of oxalates appear in the urine accompanied by increased strangury and sometimes by pain (Peyer, Ultzmann). It is thought that the oxalic crystals irritate the mucous membrane.

Some writers think that phosphaturia may be a symptom of neurasthenia. A. Freudenberg (*D. m. W.*, 1903) goes furthest in this respect, as he described, in addition to the manifest phosphaturia, a latent form, which is only evident when the urine is heated and may be recognised by the ammonia reaction—the evaporated urine staining moistened red litmus paper blue. See also Delbanco, *M. f. prakt. Dermat.*, 1904. No great value can be ascribed to this symptom. The data as to increased secretion of indican in one form of neurasthenia (Starr) are indefinite.

Trophic disorders are not prominent in neurasthenia. The most frequent are premature greyness of the hair, circumscribed canities, *e.g.* of the eyelashes, and less often the affections of the nails described on p. 65.

The *general nutrition* does not necessarily suffer in the least. Many of the patients look the picture of health. But the sleeplessness and notably the nervous dyspepsia may lead not only to pallor and emaciation, but to marked loss of strength. Progressive emaciation in spite of full feeding is much less common.

Some neurasthenics (women especially) lose and gain in weight very rapidly, showing an unusual variability in this respect.

There is, on the other hand, a form of adiposity which occurs in early youth, and is as a rule associated with a neuropathic disposition (Nothnagel, Kisch).

Some nervous children become feverish on the slightest occasion. *Fever* following mental excitement has much less frequently been reported in adult neurasthenics, but this question requires further explanation (see p. 1092).

Anæmia and chlorosis may be combined with neurasthenia, but may also be simulated by it. The results of examination of the blood require careful consideration (Gött, *M. m. W.*, 1906).

[1] *M. m. W.*, 1903.

Neurasthenia is not an uncommon affection in *children*;[1] when it appears in early childhood it is practically always congenital, but it may be acquired at a later stage. Almost every symptom, apart naturally from those of sexual neurasthenia, may occur at this age. But as masturbation may begin even in childhood, so also in rare cases may erections be a distressing symptom in the first and especially in the second childhood. The mental excitability, which takes the form chiefly of timidity and fear, gives early evidence of the neuropathic disposition. The liability of the mind to exhaustion may be shown when the child begins to study, by rapid failure of attention, exaggerated restlessness, etc. Disorders of sleep are not uncommon. Affections of the vasomotor system, *e.g.* abnormally rapid change in the fulness of the vessels, a tendency to urticaria, syncope, circumscribed œdema, etc., may be early indications of the neuropathic disposition. Gastric disturbances are not infrequent. The habitual constipation of childhood is usually due to neurasthenia. Conditions of fear and fixed ideas, hyperæsthesiæ of the special senses, and asthenopic troubles (Saenger) are also manifestations of the nervous disposition which may develop in childhood. Children are peculiarly liable to tic (see chapter on this subject), which is not, indeed, a symptom of neurasthenia, but develops on the same basis.

The symptoms of neurasthenia are so numerous that it is hardly possible to describe them in detail. It should be expressly stated that the clinical condition varies greatly in every case. Each symptom may in its turn predominate, or appear to be the only one present. Some patients complain only of pressure in the head and vertigo, others only of insomnia. In many cases the heart troubles or the sexual disorders are the chief complaints, in others the gastric symptoms. Close examination usually reveals other symptoms which are only less evident for the time being, but become marked at other periods of the disease. The distinction between cerebral, spinal, mental, and motor neurasthenia, etc., is an artificial and impracticable one.

Diagnosis.—Traumatic cases of neurasthenia (and hystero-neurasthenia) have specially led to the necessity for ascertaining the *objective signs* of this disease, and I have in my work on the traumatic neuroses pointed out many of these symptoms. Since then Loewenthal (*N. C.*, 1902) has studied the subject. Knowledge of these symptoms is rendered very desirable not only on account of the accident and compensation laws, but also of the frequency with which neurasthenia is the cause for applications for leave of absence on the part of officials, actors, etc.

Amongst the objective symptoms of neurasthenia the following may be included :—

1. *Exaggeration of the tendon reflexes* ;

2. *Exaggeration of the mechanical excitability of the muscles*, and less often of the *nerves* ;

3. *Abnormal excitability of the cardiac nervous system, palpitations*, and the other objective symptoms of *cardiac neurasthenia* ;

4. *Vasomotor, secretory, and trophic disorders*, and *spastic* and *atonic conditions* in the organs with non-striped muscles ;

[1] Literature in the section on hysteria. Other papers by E. Hirt, " A. f. Rassen," etc., 1904 ; Weygandt, " Leicht-abnorme Kinder," " Samml. zwangl. Abhandl.," 1905.

5. *Disorders of metabolism* (alimentary glycosuria, etc.);
6. *Tremor*: rapid and fibrillary.

We should not expect all these signs, or even the greater part of them to be present in every case. There are many cases in which the disease consists exclusively of subjective disorders. On the other hand the increased excitability may be directly evident in many regions, *e.g.* some slight physical exertion may produce unusual acceleration of the action of the heart and of respiration, increase of the blood-pressure, or some imaginary fear may cause an abnormally marked psycho-physical reaction. Hyperæsthesia of the retina may be shown by an abnormally lively reaction of the pupils, etc. etc. The fatigability may be demonstrated objectively by suitable psychological experimental methods (Kraepelin, Weygandt, Breukink[1]).

Patients suffering from severe forms of neurasthenia, mixed forms of neurasthenia and other neuroses, and especially from congenital neurasthenia, often present changes which should be regarded as congenital anomalies of development, *i.e. stigmata of degeneration.* Exact knowledge of all these anomalies, which are by no means sufficiently known and appreciated (a few of these are mentioned on p. 4), may be of great service in the diagnosis of these neuroses and especially in establishing the presence of neurasthenia. The stigmata usually consist in physical malformations, but may also be mental. Some psychomotor peculiarities and *congenital associated movements* have the same significance.

The achylia gastrica of Albu (*B. k. W.*, 1905), the enuresis nocturna of Oppenheim and Pfister (*M. f. P.*, xv.), and the so-called truancy of small children, of Neumann (*A. f. Kind.*, Bd. xlii.), may also be considered as stigmata of degeneration in the wider sense of the word.

I have seen chronic tachycardia in several members of one family, who suffered in addition from ichthyosis.

The diagnosis of neurasthenia should be made *by exclusion.* One must first of all make sure that no serious disease is present. There are a number of diseases which go through a preliminary neurasthenic or pseudo-neurasthenic stage. We should bear in mind that the subjective troubles felt at the commencement of phthisis have a great affinity with those of neurasthenia (palpitation of the heart, fatigue, feelings of weakness, depression, irritability, tendency to sweating, etc.). This is also true of diabetes mellitus. The symptoms of neurasthenia are sometimes practically identical with those of diabetes, and numerous cases have shown that alimentary glycosuria may be a symptom of neurasthenia.

I have sometimes found in cases with symptoms of diabetes that so sugar was present in the urine, but that there was a substance which rotated to the left (Marie-Robin, Lépine); the nature of these symptoms, which Lépine names "*le syndrome lévulosurique*," is as yet unexplained. Withdrawal of carbohydrates is said to be followed by rapid improvement. See also Hoppe-Seyler, *Med. Klinik*, 1905; Dickinson, *Lancet*, 1904.

A marked carcinoma has even been mistaken for neurasthenia. On the other hand I have treated elderly persons, including men, for nervous anorexia which had led to a condition of cachexia and thus simulated a malignant tumour of the gastro-intestinal tract. Arteriosclerosis may specially give rise to confusion, but I have very much oftener found the opposite error made of attributing neurasthenic

[1] *M. f. P.*, xv.

symptoms to arteriosclerosis, which was either not present or was not responsible for the symptoms. In many cases the diagnosis had been made and communicated to the patient by experienced clinicians, with the result that he continued for years to suffer from severe hypochondriacal neurasthenia, which was eventually cured or greatly improved by psychotherapy. I have had some of these cases under observation for ten to fifteen years, and am therefore satisfied that arteriosclerosis was not the cause of the symptoms. I cannot too strongly deprecate the misuse that some otherwise excellent physicians make of this diagnosis. The uric-acid diathesis may produce symptoms allied to those of neurasthenia, but it seems to us that too much importance is attached to this factor.

The physician is often called upon to determine whether his patient is suffering from cardiac neurasthenia or an organic disease of the heart. The presence of hypertrophy or marked dilatation is in itself evidence of a cardiac defect; murmurs which are present, not during an attack or after some excitement, but persistently, and which cannot be regarded as accidental, have the same significance, but simple accentuation of the heart's action, a vibrating apex-beat (F. Müller), increased rapidity, slightly intermittent action, a small pulse and dicrotism are all symptoms which may occur in neurasthenia. In this case they are merely temporary, whilst in organic affections they are usually chronic. Goldscheider thinks that marked arhythmia is not due to neurasthenia, but I have treated several cases of severe neurasthenia in which irregularity of the heart's action persisted for a long time, disappearing either with the improvement in the primary disease, or becoming permanent, although no other cardiac disorder developed.

F. Müller (*B k. W.*; 1906) advises that more care should be taken in the diagnosis of nervous heart diseases, as toxic and infective diseases and secondary affections of the heart muscle, such as result, *e.g.*, from myoma uteri (myoma-heart), etc., are often mistaken for cardiac neuroses.

Strong efforts have been recently made to distinguish between the arhythmia, intermittence, and bradycardia of nervous origin, and those due to organic heart-disease, the theory of Engelmann already referred to being the basis of many of these. Investigations and communications on this subject have been published by Wenckebach, Hering, Hoffmann, Knoll, Lommel, Rehfisch,[1] Reissner,[2] Goldscheider,[3] Reckzeh,[4] Hoffmann,[5] Rumpf,[6] Herz,[7] and others, and we would specially point to the papers by Kraus,[8] Goldscheider, and Krehl.[9] But on the whole the attempts to diagnose between functional myogenic and neurogenic disorders of the heart have been so far unsuccessful. Radiography and the *orthodiascopic* method (Moritz) have been employed, and they may possibly lead to important conclusions. Whether and in how far investigations with the *electrocardiogram* will aid in differential diagnosis (Kraus-Nicolai, *B. k. W.*, 1907) the future alone will show.

In cardiac neurasthenia, the pulse rate is specially accelerated by emotion, less markedly by physical exertion, although the latter may also have an undue influence upon it. Nervous affections of the heart give rise to no disturbance of compensation. They are usually associated with vasomotor disturbances. It should not be forgotten that increased pulsation of the small arteries and even a capillary pulse may be symptoms

[1] *D. m. W.*, 1903 and 1904. [2] *Z. f. kl. M.*, Bd. liii.
[3] "Diagnose des gesunden Herzens," Leuthold's "Gedenkschrift," i., and *Z. f. physik. Therap.*, x.
[4] *D. m. W.*, 1904. [5] *D. m. W.*, 1906. [6] *D. m. W.*, 1905.
[7] *D. m. W.*, 1907. [8] *D. m. W.*, 1905.
[9] Nothnagel's "Handbuch," xv., and *M. m. W.*, 1906.

of nervousness. In nervous palpitation the breathing is very rapid, although there is no *objective dyspnœa*, whilst in anginous attacks due to organic heart-disease, respiration is usually slow and accompanied by inspiratory dyspnœa due to tension of the auxiliary muscles of respiration. Digitalis has no effect in nervous affections of the heart. Finally, the general condition of the patient is of importance as an aid to diagnosis, as the symptoms of neurasthenia present indicate that the heart-disease is a nervous one, but it should be borne in mind that an organic defect in the heart may produce neurasthenia, and *vice versâ*, that a nervous affection of the heart and vascular system may develop into an organic one. It is said also that hypertrophy of the left ventricle may develop from the cardiac disorders of sexual neurasthenia, but this is exceedingly rare.

It may be specially difficult to establish a diagnosis from general paralysis, disseminated sclerosis, cerebral tumour, and cerebral syphilis or cerebro-spinal syphilis. The distinguishing features may be inferred from the descriptions of these diseases. The importance of the examination of the cerebro-spinal fluid has also been discussed there. The results of cyto-diagnosis or chemical examination of the fluid and serum (see p. 991) may establish the diagnosis between neurasthenia and general paralysis, but I have never found it necessary to resort to this method of diagnosis.

Chronic poisoning, especially the metallic forms, may lead to the development of a complicating disease, the neurasthenic troubles being associated with some symptoms of an organic disease, in particular with a neuritis. I have often seen these mixed forms, which have been described by Ziehen and Friedmann (*M. f. P.*, ix.).

Typical neurasthenia has nothing to do with *melancholia*, *paranoia*, and the psychoses of exhaustion, but mixed and transition forms occur, and the limits between them are sometimes very vague. *Hypochondria* is specially apt to accompany neurasthenia, and their intimate relation is recognised by most writers.[1] Redlich regards the condition known as hypochondria as a further development and exaggeration of the symptoms of neurasthenia. A combination of neurasthenia with melancholia, or a form of neurasthenia in which the depression for a time very nearly approaches in its intensity and character the melancholic form, is not a rare occurrence. It may be very difficult to distinguish congenital neurasthenia, especially its degenerative, psychasthenic forms, from *moral insanity*, as the abnormalities of psychasthenic children may give the impression of ethical inferiority. Prolonged observation and critical consideration of the incriminating actions will usually lead to a decision, as it can be recognised whether they are manifestations of imperative impulses or of a moral defect. The boundaries between degenerative neurasthenia, psychopathic inferiority, and moral insanity are not always clearly defined. *Dementia præcox* may in its commencement give rise to symptoms allied to those of hysteria and neurasthenia. The differential indications will be found in text-books on psychiatry. Personally I would merely advise that the diagnosis of dementia præcox should be given with reserve, as I have repeatedly been afraid that this disease was about to develop in cases which have been shown by their further course to be those of a form of neurasthenia or hystero-neurasthenia.

[1] Compare on the subject Wollenberg, "Hypochrondrie," in Nothnagel's "Handbuch," xii., and *Z. f. N.*, 1905.

See also Birnbaum's essay upon degenerative eccentricity (*N. C.*, 1906). Some forms of manic-depressive insanity (Kraepelin) are so slightly developed that they may be mistaken for neurasthenia. Finally, I have had under my care some patients with very marked heredity whose condition contained the elements of neurasthenia, hysteria, hypochondria, and even of melancholia.

The limits between *neurasthenia* and *hysteria* are not sharply defined, as these neuroses are often associated with each other and have many common symptoms. Nevertheless, they are by no means identical. Although hysteria is often accompanied by neurasthenic symptoms, neurasthenia very frequently occurs in its pure form. In neurasthenia the *spasm*,[1] the *paralysis*, the *anæsthesia*, and the *anæsthesia of the special senses* are absent. The abrupt changes of mood, the sudden onset and disappearance of the symptoms, do not belong to neurasthenia. The symptoms which tend to appear in paroxysms, *e.g.* palpitation and vertigo, are transient in nature, but the primary disease is characterised by the constancy of the symptoms, although they vary in their intensity. Exaggeration of the tendon reflexes and of the mechanical excitability of the nerves and muscles, fine tremor, fibrillary twitching, and urticaria factitia are neurasthenic symptoms, but they are often present in hysteria, in which case they represent either a neurasthenic accompaniment of the hysteria or are sufficiently characterised by their psychogenic origin. Hysteria is specially distinguished by exaggerated suggestibility. In neurasthenia the chief feature is the fatigability of the nervous system. Morbid introspection is certainly a factor in neurasthenia also ; it creates fresh troubles and increases those already present, but it does not give rise to paralysis, anæsthesia, or contracture, nor do the symptoms and syndromes for which it is responsible appear and disappear as if by magic.

Chronic headache is usually a symptom of neurasthenia, but there are cases and forms which show no other sign of this neurosis. Charcot, for instance, would distinguish between cephalæa adolescentium and neurasthenia.

The conditions most nearly approaching neurasthenia or hystero-neurasthenia have been described by Bornstein (*N. C.*, 1905) as asthenia paroxysmalis, but further study is required to explain them.

Anorexia, like many other symptoms, may develop on a *neurasthenic* as on a *hysterical* and *psychasthenic* basis.

Nature of the Disease.—We believe with Bouveret and others that neurasthenia is a disease of the nervous system as a whole, and is caused by changes in the nervous elements so minute that our present methods of investigation fail to detect them. Although Nissl's method has revealed structural change in the nerve-cells in various conditions of chronic intoxications associated with neurasthenia, it is still doubtful whether the pathological basis of this condition has been discovered. The many theories which have been put forward as to the nature of neurasthenia cannot be discussed, but the most important points

[1] This sentence requires limitation. According to our experience, attacks of loss of consciousness with or without convulsions may appear in neurasthenia, being due to the neuropathic and psychopathic diathesis. This is specially the case in the vasomotor and degenerative forms of neurasthenia. The attacks differ, however, from those of hysteria and are more nearly allied to syncopal and epileptic attacks. See my paper upon psychasthenic spasms, *Journ. f. P.*, vi. ; also Friedmann, *Z. f. N.*, xxx. ; Spiller, *Journ. Abnorm. Psychol.*, 1907, and the chapter upon epilepsy in this textbook.

have already been referred to. We have spoken in the previous chapter of the attempt to relate neurasthenia and hysteria to disorders of metabolism (Vigouroux, Biernacki, Higier, Haig). Changes of this kind—in particular increase of the secretion of uric acid, etc.—have been reported by Huchard, Bechterew, and others, but it is very doubtful whether they are the cause of neurasthenia.

Course and Prognosis.—The onset of the disease is usually gradual, but in a few instances it is acute or even sudden. As a rule it has a chronic course, increasing gradually from year to year or becoming arrested after a longer or shorter interval. It generally lasts for several years, decades, or even for the whole lifetime, although it is not of uniform severity throughout, but is broken up by remissions and complete intermissions. Exacerbations of the mental symptoms may take the form of paroxysms or crises (Diehl), particularly in the degenerative forms. See the appendix.

There is sometimes a certain periodicity in the coming and going of the neurasthenic attacks, but we do not think Sollier,[1] Lange, Hoche, Dunin[2] and others are justified in speaking of a circular form of neurasthenia. I agree with Pilcz, Redlich, and others that the cases included in this category are practically always those of circular insanity, which in their external aspect resemble the neurosis rather than the psychosis.

The stronger the constitution of the patient, the less marked his nervous heredity or neuropathic diathesis, and the less advanced the disease, the better is the *prognosis*. It is also more favourable in cases that develop acutely than in those which are chronic from birth, and better in acquired than in hereditary neurasthenia. A combination of the latter with signs of mental degeneration makes the prognosis very grave. Raymond in this connection makes an important distinction between neurasthenia and psychasthenia. So far as the prognosis depends upon the cause, it appears to be most favourable when the disease is due to mental exhaustion. It often depends to a great degree upon the patient's social position and the possibility of attaining the conditions suitable for his recovery. Complete recovery is often attained, but it is less likely to occur if the disease has lasted for many years. But even a marked improvement is possible at any stage. In those that do not recover, the danger is not great. It is only cases with a very marked hypochondriacal tendency or mental deficiency that develop into psychoses. Neurasthenia practically never precedes an organic nervous disease. One point must be kept in mind, namely, that when the cardiac nervous system is involved, there is always a fear that prolonged excitement of the heart and vascular system, with constantly recurring acceleration of the action of the heart, may lead to an organic disease in that organ. I have very often found *atheromatosis of the heart and vessels*[3] develop at a comparatively early age in such cases. This neuro-vascular origin of arteriosclerosis is not common, and possibly it may be due in part to congenital weakness of the vascular system, which leads to its being worn out early (Oppenheim). Goldscheider also thinks that prolonged nervous dis-

[1] *Rev. de Méd.*, 1893. [2] *Z. f. N.*, xiii.

[3] I have stated these facts in my first articles upon the traumatic neuroses. Runge found tortuosity and dilatation of the temporal artery on the affected side in persons who had for a long time suffered from unilateral congestion of the head. Thoma and his pupils have confirmed these relations by pathological investigations. Régis, Fraenkel, Romberg, Goldscheider (*B. k. W.*, 1906), L. R. Müller (*M. m. W.*, 1906), and others have published communications on this subject.

orders of the heart affect that organ, that the nervous heart has less power of resistance, etc. Neurasthenia may thus prove indirectly fatal.

In very rare and severe cases, the sleeplessness and especially the condition of anxiety, may drive the patient to suicide. In such cases there is some mental disease, with hypochondriacal or hypochondriac-melancholic paroxysms, but it may be difficult to determine at what point the neurosis develops into the psychosis. The fear of mental disease may produce a condition of great anxiety and lead to attempts at suicide. The " transitory agrypnic delirium " described by Agostini, *i.e.* a mental disturbance of acute onset due to persistent insomnia, is exceedingly rare.

The *treatment* of neurasthenia may be a very gratifying but at the same time a very difficult task. In many respects it is identical with that of hysteria described in the preceding chapter.

Medical advice and care may do much to ward off the disease, but *prophylactic* measures lie for the most part beyond our province. They include a return to the simple life, to an earlier state of civilisation, depopulation of our large cities, a war against the desire of money, prevention of marriage between relatives, degenerates, etc. These, and many other factors which make for the hygiene of the nervous system, are unfortunately seldom the object of medical care. The family physician, however, is in a position to use his influence in favour of the rational education of the growing generation, especially of children with a nervous tendency; to urge early measures for counteracting the disease, and for shielding the child from injurious influences. Everything which strengthens and hardens the young physique forms a protection for the nervous system. Everything which oppresses the mind, arouses the senses, excites the imagination, and softens the body prepares the way for neurasthenia (and hysteria). The inferences to be drawn as regards education are self-evident (see p. 1101). Care is necessary in the *choice of an occupation*. The individuality should be fully considered, and the nervous disposition kept in view. When the nervous tendency is marked, every effort should be made to avoid choosing an occupation which demands high responsibility and activity associated with great excitement. Backward children should not be forced to study. One cannot lay down hard and fast rules in this respect, as natural capacity, social position, and individual preference are all-important points to be considered.

A. Hoffmann has written a monograph on this subject: " Berufswahl und Nervenleiden," Wiesbaden, 1904.

It is very important that nervous children should be prevented from masturbation. We do not know any infallible remedy, but if the child is carefully watched, brought up to be frank, and talked to openly at the right time, he will be much less likely to acquire the habit.

When the disease has fully developed, treatment must first be directed to the cause. If there is mental exhaustion, all activity must be stopped. The patient should not, however, be condemned to absolute idleness; mental work should be replaced by physical, or the patient should be ordered to avoid exhaustion by resting frequently for at least some hours from his work. In any case the physician should not (unless under special conditions) advise a man who has hitherto been capable of working to give up his business entirely. I have very often found the nervousness

greatly increase or first appear from the time when the patient retired from business, as his unoccupied attention turned directly in upon himself. Work which is adapted and rightly proportioned to the patient's capabilities is on the contrary an invaluable remedy. On the other hand, in the grave forms of neurasthenia characterised by exhaustion, the only beneficial method of treatment is that of complete rest and absence of all sensory excitement.

The *causal indication* may call for the treatment of a nasal affection, a disease of the sexual system or the stomach, the removal of tape-worm, etc. It should be remembered that gastric troubles are as a rule the outcome of nervousness, and that sexual disorders are often not a cause, but a symptom of neurasthenia. If possible the patient should be cured of masturbation. The physician must judge how far the patient should be made aware of its dangers. The hypochondriac, haunted by the fear of a disease of the brain or spine, should not of course be further alarmed. The evil done in this respect by published accounts of the results of onanism can hardly be estimated. In other cases it is necessary to point out the dangers and the possible consequences of masturbation and to use one's medical authority to encourage the patient's feeble will and to urge him to continence. A special warning should be given against *psychical onanism*. It may perhaps be necessary to remove the patient from his actual environment. The best protection against masturbation and its results is *physical exercise*, much activity in the open air, gymnastics, climbing, swimming, fencing, or a régime like that of military service, etc. Possibly hard manual or farm labour from morning till night may be advisable. Bicycling is of great service to many neurasthenics.

These are at the same time excellent remedies for emissions and sexual neurasthenia. It may be well to occupy the mind with hard mental work, to encourage the study of natural science, photography, preparation of microscopic slides (Vogt), and the numerous impressions gained from travel in a foreign country, a sea-voyage, etc. Much harm is done by local treatment of the urethra with drugs, and Lallemand's caustic carrier and similar apparatus should be strictly avoided. Even when chronic gonorrhœa is present, these measures have often an unfavourable effect upon the general condition, increasing the nervousness and with it the sexual disorders. Fürbringer advocates the use of Winternitz's double-current irrigator (the psychrophor), especially in cases of hyperæsthesia of the prostatic region. Water of 14 to 8° C. should be applied for a considerable time (up to ten minutes) to the urethra by means of the special catheter-like instrument. Winternitz himself found this successful in emissions, spermatorrhœa, and chronic gonorrhœa. Impotence due to these causes is an indication for this treatment. The *general treatment* of the neurasthenia, according to the methods to be described, is always the most important point. No real benefit is to be gained from the use of so-called pollution rings and similar " wakeners," or from genital protectors. A physician who consulted me for this disease had invented many wonderful contrivances by which he was awakened by each erection. It may be advisable to prevent contact of the bedclothes with the genitals by means of a wire frame, etc., as voluptuous sensations and emissions are often caused by tactile stimuli. The treatment of masturbation by means of painful applications to the penis (collodium cantharidat, etc.) is of no permanent benefit. *Electrotherapy* is some-

times very useful in sexual neurasthenia. For emissions and spermatorrhœa I use the galvanic current, a large cathode being placed upon the lumbo-sacral region, and a stabile anode of about 20 sq. cm. upon the perineal, testicular, or lumbar region; with a current of 4 to 6 M.A. Intra-urethral faradisation may also do good. *Lupulin* is recommended for emissions, but I have found *camphor. monobromat* (½-3 grs.) more useful. Heroin has been recently recommended. I have often been successful in treating neurasthenic impotence with the faradic brush, applied especially to the testicles, perineal and lumbar regions. Galvanic treatment in which an electrode is introduced into the rectum, and faradisation of the prostate from the rectum (Porosz), have also been advocated. A cold douche applied to the lumbar and sacral region may do good, and so may carbonic-acid baths. Cathelin's method, said by a few physicians to be useful, will, no doubt, soon be abandoned. I attach no value to such drugs as cantharides, strychnine, phosphorus, etc., in impotence. In slighter cases spermin seems sometimes to be useful, but this is due merely to its suggestive and perhaps its tonic effect. During the last ten years Oberwarth-Spiegel's *yohimbin* (5 to 10 drops of 1 per cent. solution yohimbini Spiegel, or tablets containing 5 mg.) has been adopted, but it was badly borne by some of my patients and was of little use. Fürbringer[1] and others have found the same result. I am also unable to report favourably of the drugs recommended recently as aphrodisiacs.

At an earlier period I was somewhat opposed to the method recommended by Zabludowski (*Z. f. physik. Ther.*, iii. and x.) of producing artificial hyperæmia of the penis by means of the air-pump, etc., but I must admit that it has had beneficial effect upon a few cases under my own observation.

The apparatus (erector, etc.) which have been advertised for stiffening and extension of the penis have proved of no use in my cases. Local massage, which has also been recommended, may, I think, do more harm than good.

It is often necessary to conceal mental treatment behind some indifferent drug. Neurasthenics who in marrying feel timid concerning the first attempts at cohabitation, may be helped over the anxious period, and sometimes even permanently cured, by the prescription of some indifferent remedy and the assurance that it will have the desired effect.

It may be a very delicate matter for the physician to decide whether a patient suffering from sexual neurasthenia ought to marry or not. So far as my own experience goes, marriage has a very beneficial effect in the great majority of cases and leads to full recovery of the diminished sexual power. Should there be absolute impotence or perversion of sexual desire, the physician should not take the responsibility of encouraging marriage.

Hypnotic treatment may help to counteract perverse tendencies (homosexualism, etc.), but, as Bernheim states, it is usually of no avail. Some hypochrondriacs and psychasthenics believe wrongly that they have contra-sexual tendencies, and in such cases careful psycho-analysis may be required to enable the physician to recognise the condition and treat it in the right way.

Treatment of *nocturnal eneuresis* consists in removal of the cause, which may be of reflex origin (phimosis, intestinal worms, vulvitis, adenoids), in prescribing a dry evening meal, wakening the patient at certain hours during the night, keeping the pelvis in a raised position, and psychotherapy. Troemner, Cullère, and Delius (*W. kl. R.*, 1906) have found hypnotic treatment

[1] See the latest work on this subject in the *D. m. W.*, 1907; also Posner, *Ther. d. Geg.*, 1907.

successful. It is suggested to the patient that he will be aware of the pressure and waken from it. Very little can be expected of the drugs, viz. belladonna, atropin, bromide of potassium, tinct. rhois aromatica, in ten to fifteen drops, etc., which have been recommended. Seeligmüller and Köster have advised internal faradisation, the electrode being introduced into the urethra for a distance of one to two centimetres and a painful current being employed. This method may be tried. The introduction of an electrode into the rectum has also been found useful. Combined massage (from without and from the rectum) is recommended by Walko. I do not think it advisable to use metal catheters, still less to cauterise the neck of the bladder, etc. Good results have been reported in some obstinate cases from Cathelin's method (Kapsammer, *W. kl. W.*, 1903, etc.), but nothing has been said with regard to it during the last few years. As a rule the disturbance passes off spontaneously and rarely persists after the age of puberty.

As regards the *general treatment* of neurasthenia, drugs are of little service. In many cases they need not be given at all. It is often necessary and advisable to give sedative medicines, such as the various *preparations of bromide* (bromide salts, Erlenmeyer's bromine water, bromipin, bromalin, quinine-hydrobromide). One should, however, make it a rule only to prescribe these drugs for a short time, perhaps for a few weeks, or for a longer period only when small doses, given at long intervals, have a material effect. We should never forget, when prescribing drugs, that nervous patients are *extremely* sensitive to them, so that it is often necessary to discover by repeated trials the actual dose which will produce the desired effect. Although suggestion certainly plays a great part in this, I do not believe that the differences of effect and sensitiveness can be ascribed to it alone, as the reaction of many neurasthenics, even when in good health, to alcohol, nicotine, etc., shows how greatly the sensitiveness for toxic substances may be exaggerated. I believe *arsenic* to be a drug of great value. It should always be tried in cases of severe and chronic neurasthenia. In addition to the internal use of drugs, subcutaneous (or intramuscular) injections of various preparations (atoxyl in doses of $\frac{3}{4}$-$1\frac{1}{2}$ grs., cacodylate of soda, etc.) have been recommended. If anæmia is present, iron may also be given, but the anæmia of neurasthenics is often secondary or merely apparent, and in my opinion too many experiments are made with preparations of iron. Preparations of phosphorus (phosphate of soda and glycerophosphate of soda), lecithin, and a combination of iron and strychnine, or phosphorus and zinc, have been recommended. The preparations which I need not name, containing a number of these drugs in solution, may also be prescribed. The other nerve tonics may benefit the various symptoms, but one cannot be too careful in prescribing antipyrin, phenacetin, antifebrin, analgen, citrophen, etc., and in any case they are not adapted for continuous use.

I cannot agree with Kowalewski's view that neurasthenia may be due to syphilis, and curable by specific treatment.

Psychotherapy [1] has a wide field in neurasthenia. It is the *chief factor in the treatment* of many of the symptoms, and it may be advisable to increase the effect of suggestive influence by the addition of hypnotism. No valuable results can be obtained without a proper knowledge of the nature of the disease and of the patient. On the other hand the influence of medical encouragement, based upon an exact knowledge of the disease and a careful examination of the patient, is in most cases wonderfully helpful. In some cases the patient requires nothing of his physician except the confident assurance that he is not suffering from a grave

[1] See the bibliography in the chapter on hysteria.

organic disease or a psychosis. I know a number of neurasthenics who consult me regularly at long intervals simply to be re-assured upon this point.

In severe cases and forms the personal influence has usually to be exerted continuously and for a long time. The physician must, so to say, attach himself to his patient in order to guide him aright and carry him over the difficulties in his path.

Hydrotherapy is one of the most valuable methods of treatment. *Cold rubbings*, *lukewarm* or *cold half-baths*, *sitz-baths*, *sprays*, and *douches* are specially good. The cold rubbings are well borne by the majority of patients, if they are begun with a temperature of 25-27° R. (88-92° F.). If the patient is too sensitive even for this, partial rubbings may be beneficial. In delicate, anæmic, or very sensitive cases it is best to begin rubbing with alcohol and to avoid the effect of cold for a time. The treatment can usually be modified so as to suit the patient. An energetic method, which may be useful in robust cases, is to precede the cold rubbing by washing with hot water or even with hot salt water. This procedure may be adapted to delicate persons if it is limited to one limb at a time. Sprays and douches, and pouring water over the patient are only suitable for cases with a greater power of resistance. Douching of the head should be avoided, but lukewarm sprays can usually be borne even by weaklier patients. *Wet packs* have a soothing effect and usually induce sleep. Cool or cold baths of 25-20° C. (77-68° F.), lasting only for some seconds, have often a soothing effect in conditions of excitement, and when the patient is relaxed generally, hot full baths of short duration may have a strengthening and rousing effect. Cold sitz-baths of 15° R. (66° F.) or short, or lukewarm, or gradually cooled baths of long duration are often beneficial. The individuality of the patient is the most important factor in all these respects.

All these methods may be carried out at home, but, *ceteris paribus*, the result is better if the patient is heated in a well-conducted *hydropathic establishment* under the supervision of a trained neurologist. The fact that residence in an institution avoids a number of excitements which the patient might have in his own home contributes to this good result. On the other hand great harm is done by neurotic persons meeting in sanatoriums and spreading the *mental infection* by constant conversation upon and comparison of their complaints. Those in charge of such institutions should absolutely forbid any conversation bearing upon the maladies of the patients. Prohibition is not of course of much use. We have already, in the chapter upon hysteria, pointed out the value of institutions in which treatment of the nervous condition is associated with *occupation*.

In many sanatoria, sun and air baths are thought to be important factors in treatment, and their value should not be under-estimated. On this subject see Determann in Goldscheider-Jacob's "Handbuch. d. physik. Therapie." Treatment by *rest in the open air* has often an excellent effect, particularly in those forms of neurasthenia associated with poor nutrition.

Residence at the sea-side has sometimes (though in a minority of cases) a beneficial effect, but its effect can never be absolutely predicted. Cold sea-bathing can only be borne by robust neurasthenics and in the early stages of the illness, but with all these prescriptions one must be prepared for all kinds of surprises and consider very carefully the condition of the

heart and vascular system. The baths on the Baltic coast are less exciting and vigorous than those of the North Sea. Some of the symptoms, such as the neurasthenic headache and insomnia, may be helped by a long sea voyage. *River baths* and *cool brine baths* have led to marked improvement in some cases. The indifferent baths of Gastein, Schlangenbad, Johannisbad, Landeck, etc., have occasionally been useful, and also the iron waters of Pyrmont, Elster, Franzensbad, Schwalbach, Kudowa, etc.

Climatic treatment may be of great value. Mere change of scene has generally a good effect, but there are exceptions to this rule. We cannot here discuss the special indications for the selection of a place in the country, in the woods, for the high or lower mountains, for the seaside or a sea voyage ; we have as yet no reliable and well-proved data gained by observation and experience. It may be said, however, that high mountains are not generally suited for excitable and debilitated neurasthenics nor for those suffering from commencing or declared arterio-sclerosis. Nevertheless I know some people of advanced age, who have spent the hottest part of the summer in Pontresina or some similar place, and have there enjoyed the best of health. Residence in the mountains is contra-indicated, according to Krafft-Ebing, by conditions of anxiety, and Loewenfeld thinks also by cardiac and vasomotor neurasthenia. But a few cases have been described in which affections, such as agoraphobia, have recovered in the mountains. Further, it has to be remembered that patients who require medical supervision can only find it in certain climatic resorts, most readily in institutions for the treatment of nervous diseases.

Although the German climatic resorts are specially adapted for visits during the summer months, the patient may derive great benefit from being sent south during the cold weather, *e.g.* to the Mediterranean coast. Wintering in Heligoland, St Moritz, etc., may have a very good effect, as Erb[1] and Laquer have specially stated. The German hill-resorts, such as Schierke and Oberhof, are well adapted for this purpose, especially as the winter sports, which are so health-giving, may be had there.

Electrotherapy finds a wide and fruitful field of activity in this affection. *Galvanisation of the head and back, general faradisation, Franklinisation and electrical baths*, especially the four-cell bath, have been much advised, and have in our experience proved very helpful. In addition to these the Tesla-current, the magneto-electrical method, the high frequency currents, and the monodic volta current have come lately into use. The results of electric treatment can by no means always be predicted ; in every case we have to experiment, but if we go carefully forward, commencing always with the weakest current, we need not be afraid of the results.

Leduc's method has not yet been sufficiently tried. See Mann, *Z. f. med. Elektr.*, 1907.

General massage, active and *passive gymnastics*, and *medico-mechanical* treatment are specially suitable for listless and inactive patients, who cannot rouse themselves to physical exertion without some corresponding object. It is also a powerful factor in the psychotherapy of other cases, either from its effect upon pain or its power of diverting the patient's attention. Zander's apparatus, Gaertner's ergostat, the apparatus for

[1] See his latest papers in *Ther. d. Geg.*, 1907.

practising rowing, the bicycle for home exercises, etc., may all be suitable for this purpose.

As regards the *diet*, sameness in the food should be avoided, and too much importance should not be attached to meat as a constituent. The food should be mixed and easily digested. The meals should be small and frequent rather than large and few, and the diet should be adapted to the condition of the patient's nutrition. When there is a marked "uric-acid diathesis," the dietary should be to a certain extent modified accordingly, but the importance of this factor has been greatly over-estimated by Haig [1] and others. We cannot be too emphatic in advising the avoidance of *forced feeding*. Some of the worst cases of neurasthenia which I have seen have developed as the direct result of a so-called "Schweningerkur." Alcohol should be limited as far as possible, and the idea of many neurasthenics that they should drink brandy must never be encouraged. On the other hand, I do not agree with those who say that very small quantities of alcohol are always contra-indicated.

The treatment of some of the specially common and intractable symptoms will be indicated in an appendix.

As regards treatment of neurasthenic headache, see the chapter on cephalalgia.

Insomnia demands a regular mode of life: the patient should go regularly and early to bed, with little in his stomach; he should avoid mental work and all excitement before going to bed, and should during the day lead an active, open-air life. When this is not sufficient, a lukewarm bath for a half to a whole hour, a foot-bath of cold or hot water or water of varying temperature, cold water poured over the feet, a short lukewarm spray bath of 35-30° C. (95-86° F.), or a wet pack before bedtime may be useful. In other cases general massage, massage or galvanisation of the head, general faradisation, an electric (faradic) bath, the Arsonval currents, which Kindler, Bädeker, and Nagelschmidt among others have found useful, and vibration massage have the desired effect. A change of air is often sufficient to restore the power of sleep, and a stay amongst the wooded hills is specially good. Residence at the seaside, with the wading or paddling on the wet sands for which there are special facilities at Büsum, for instance, may have a soporific effect, though that can never be predicted. In advising changes of this kind it is well to take into account the patient's previous personal experience. Gastein has often a favourable effect upon sleepless neurasthenics; the ceaseless murmuring of its waterfalls has at first a disquieting effect in some cases, but this as a rule soon wears off and the sound becomes soothing. Wet packs to the whole body, Priesnitz's bandages about the abdomen, feet, etc., may induce sleep. The great benefit of hydrotherapy in insomnia is universally recognised. Psychotherapy is also of the greatest value, especially when the physician can talk soothingly to his patient just before the time for sleeping. When this kind of mental influence fails, hypnotic treatment is often successful.

If it is necessary to prescribe drugs, preparations of bromide, such as bromine water, should be tried first. Hypnotics should be avoided to begin with. *Paraldehyde* in doses of 50-90 m. is a good soporific; it is on the whole very quick and certain in its working, but has a very objectionable taste (it is given in sweet tea or claret). *Sulphonal* (15-25 grs.) is also

[1] "Harnsäure als ein Faktor bei der Entstehung von Krankheiten," Berlin, 1902.

good, but it cannot be used for a long time as it may produce symptoms of poisoning, such as vertigo, ataxia, hæmatoporphyrinuria, myelitic symptoms, etc. I regard *trional*, in doses of 15-25 grs.—I usually give 18 grs.—and veronal (5-8-15 grs.) as the most successful and least dangerous narcotics. The latter especially is, I think, the sovereign drug for inducing sleep. No ill effect can be anticipated from a single dose, if it is of the usual strength, and even from its continued use, if it is taken with interruptions. I have met with no case of severe veronal poisoning. I have known some persons who have taken it for years with no bad effect, although in others the health was disturbed, or an attempt to withdraw it was followed by symptoms which could only be attributed to the withdrawal. I have to admit, however, that it completely failed in a few cases. It is also a drawback that its soporific effect is comparatively late in appearing. *Proponal* is in my experience a very useful drug, but the ordinary dose should not exceed 3-5 grs., in order to avoid ill effects. Its effect is somewhat less certain and marked than that of veronal. Sulphonal should be given in some hot drink (milk, tea, soup) about an hour before bedtime. It is well to accompany it by some alkaline salt or water. Trional should be given in the same way, but it is more rapid in its action. It is advisable to let the solution boil for a few minutes. Veronal may be prescribed in the same way, but not in tablets. Small doses of trional and paraldehyde may be combined or given as suppositories. Trional may also be used in the form of carbonic-acid trional water. Amyl-hydrate (30-40 m. or more) and dormiol in doses of 7½-15-25 grs. are useful narcotics. None of these drugs should be taken for any length of time. 15-30 grs. of hedonal (given in wafer-paper with very little, if any, fluid) is harmless, but is much less certain in its effect than trional. Little can be said in favour of the other narcotic drugs. Morphia and chloral-hydrate can usually be dispensed with. But whenever it is necessary to procure for the patient a short period of sound sleep and to convince him of his power to sleep, there is no objection to the temporary use of these drugs. In such cases hyoscin in doses of one-third of a milligram may be good, especially in conditions of violent excitement. One must not depend upon the rare cases in which chloral-hydrate has been used for years without any bad effect. In old people (and in those suffering from diseases of the heart, lungs, kidneys, and from gout) it is better to abstain entirely from this drug and to be very careful in the use of trional and sulphonal. Chloral-amid (30-45 grs.) may also be mentioned. We should discourage the use of chloralose (1½-5 grs.). I have little experience of isopral. Neuronal (7½-15 grs.) and bromural (5-10 grs.) may be recommended for their soothing effect, but they have a less marked and certain power of inducing sleep.

Nervous dyspepsia often yields to the methods recommended for treatment of the general disease, especially to hydrotherapy, electrotherapy, change of climate, and not least to psychotherapy. Fürbringer and others have pointed out the beneficial effect of cycling, and I have known many cases in which the nervous dyspepsia has been cured when the patient, who has hitherto led a sedentary life, is induced to take up gymnastics, rowing, cycling, or riding, which give him exercise in the open air and distract his attention from himself. The Weir-Mitchell method, and Playfair's amplification of it are often very successful in the severe cases which show more or less marked emaciation on account of deficient nourishment. The Weir-Mitchell method, when strictly

carried out, consists in isolation, rest in bed, massage, electricity, and overfeeding.

The patient should be treated in a suitable hospital or sanatorium. All visits should be prohibited, and he should be watched by a trained nurse to prevent any attempt to move his muscles, or any mental excitement. He should not be allowed even to feed himself. At first he is given only milk, 100-120 c.c. every 2 hours, the quantity being increased after 3 days until he is taking 2 to 3 litres within the 24 hours. After 4 to 8 days some bread and butter and an egg, and then light meat may be added to the dietary, until after 10 to 14 days he is taking three full meals and 1 to 2 litres of milk. Malt extract, wine, and beef-steak should be added. In other cases, instead of a pure milk diet, soup (made with oatmeal, barley meal, fine flour, farina) may be given from the first in addition to the milk. Somatose has also been useful. As there is no active movement, metabolism and assimilation will be assisted by the patient being massaged daily, at first for half an hour, later for one to two hours, and faradic stimulation may be applied to the muscles for forty minutes every day. Towards the end of the treatment the massage may be replaced by passive and then by active gymnastics.

This treatment should be continued for six to eight weeks. It is contra-indicated by any abnormal mental condition, especially by melancholia and paranoia. The method may be *modified* in many ways, and a mild rest cure, in which the chief elements are sufficient rest, the patient lying in bed or on a couch for three to six hours every day, and forced feeding, is an excellent measure in cases of debility and neurasthenia. In common with other neurologists, I hardly ever prescribe the Weir-Mitchell method in its strict sense, but I have used it in a modified form with good results, even in cases in which an increase in weight and better nutrition were not the most pressing matters. When the conditions are favourable, the home comfortable, and the relatives judicious, I permit the treatment to be carried on at home.

As regards the treatment of constipation, consult the previous chapter. As a rule it should be counteracted, not by aperients, but by a change in diet and in the mode of life as already described. Honey, whole meal bread, kefir, milk-sugar, pure butter by the tea- or tablespoonful (Ebstein), cider, fruit (taken on an empty stomach) are specially useful. The milk-sugar should be added to milk and other fluids in large quantities—a tablespoonful three to four times a day according to Boas. In obstinate cases it may be well to order a purely vegetable diet for some time. Noorden and Dapper have further developed the principle of intestinal gymnastics by feeding on gritty food. It has already been pointed out that massage of the abdomen may be very useful, especially in cases of intestinal atony. Gymnastic exercises to the abdominal muscles (consisting in active flexion of the trunk against resistance in the recumbent position) may strengthen the muscles for abdominal compression and thus counteract the constipation. Cold-water enemata, glycerine suppositories, irrigation with olive oil, oil of poppies, etc., are useful remedies, but they can only be employed for a time. Constipation due to spastic conditions of the intestinal muscles should be treated by warm applications, Priessnitz's packs, warm douches, etc., and bromide, valerian, and possibly opium given internally. In any case drastic medicines should be avoided, but it is often advisable to prescribe some drug which will stimulate intestinal action mildly. Rhubarb, tamarind, magnesia, preparations of sulphur, cascara sagrada, regulin (1-2 tablespoonfuls), etc., may be given. I have found *manna* (cassia fistulosa) very useful.

I have found colombo the most useful of all the general and local methods of treating neurasthenia diarrhœa. Ziehen recommends calc. carb. 15 grs., calc. phosph. 15 grs., bismuth. subnitr. 8 grs., etc. For the treatment of hyperacidity, see Boas, *Therap. Mon.*, 1906.

Nervous *palpitation of the heart* requires above everything mental treatment. The patient must be assured that his heart is not affected, that the palpitation is of no importance, and that it is brought on and intensified by his own observation. Mental distraction during the attack, or even a quiet walk or drive, may rapidly arrest the palpitation. Cold compresses or a mustard plaster in the region of the heart have often a soothing effect. Whenever it is possible, however, it is better not to employ these remedies, which are also used in organic heart-disease, and to rely solely upon mental influence. Tapottement of the back may do good. Bromide of sodium is the best drug. Rosenbach recommends chloral-hydrate in small doses (15 grs. in 45 m., aq. dest., ten to twenty drops in half a wineglassful of water). Breuer found diuretin of service in angina pectoris due to spasm of the coronary arteries. Compression of the vagus is said to check the attack of proxysmal tachycardia (H. Schlesinger).

In cases of marked cardiac neurasthenia, general treatment is the first requisite, and hydrotherapy is very valuable. Winternitz specially recommends the application of cold to the back and neck. Cold bathing of different parts of the body, cold sprays, active and passive gymnastics, etc., often have a palliative and curative effect. Carbonic-acid baths are often very beneficial. I have not found that nervous heart troubles are benefited by the use of apparatus or by pads of wool (pelottes) for supporting the heart. For psychotherapeutic reasons I would avoid sending patients to Nauheim, but as some of them, especially those who come from Russia, are firmly convinced of the association between Nauheim and heart-affections, it is sometimes impossible to persist in refusing to send them there. Cardiac neurasthenia is not a counter-indication for sea-bathing. If there are any signs of heart weakness, or any doubt as to the diagnosis, it is wise to abstain from energetic or uncertain methods of treatment.

Nervous *asthma* calls for the removal of the exciting causes, which should always be discovered, and consist in climatic conditions, pollen from certain flowers, agitation, etc. According to Mackenzie, Fränkel, Daly, and others, treatment of a nasal condition, in particular removal of polypi and growths of the mucous membrane, may be of great service. If the so-called asthma-points are found in the mucous membrane of the nose, it may often do good to paint them with cocaine. Many cases are cured by pneumatotherapy. The general treatment of the condition (hydrotherapy, electrotherapy) is the most important element of cure, and it again is subject to the influence of psychotherapy. The importance of the latter is emphasised by Brügelmann, and I have seen cases cured by mental treatment alone. Among the so-called asthma remedies we can merely mention iodide of potassium, quinine, arsenic, tinct. lobeliæ, atropin (Riegel). The latter is specially adapted for use during the attack itself, and preparations of stramonium (*e.g.* in cigarettes), amylnitrite, nitre-paper, and in severe cases morphia are recommended for the same purpose. Sihle advises a combination of digitalis, iodide of potassium, and heroin. Pyrenol (8-15 grs.) is one of the latest remedies. Bathing the hands and feet in hot water may arrest the attack (Hoffmann, Oppenheim). Strüm-

pell (*Med. Klinik*, 1908) finds the electric light bath of much service. Inhalation of oxygen, recommended by Michaeli, is often soothing in its effect.

As to the treatment of conditions of anxiety, consult the following chapter. Neurasthenic noises in the ear are sometimes alleviated by the constant current (the anode on the side of the affected ear, with a weak current, slowly opened and closed). Preparations of bromide may also lessen the intensity of this symptom. Cimicifuga racemosa (extr. in daily doses up to thirty drops) is not to be depended upon. Distraction is the most important factor. The patient must be persuaded to pay no attention to the noises, to think of something else, and when he does so the symptom will lose its disquieting and distressing character.

For climacteric symptoms Landau recommends the use of ovarian extract from cows and pigs, given in the form of tablets (see p. 1103). Cold baths and compresses, menthol-cocaine ointment, suppositories of cocaine, eucaine ($\frac{1}{3}$ gr.), dionine, etc., and lactic acid given internally in doses of 6 to 20 drops (Du Castel) may be prescribed for local *pruritus*. Eulenburg and also Nagelschmidt (*D. m. W.*, 1907) found local application of Arsonval's current useful in such cases. One of my patients who suffered from obstinate pruritus ani always found great relief from anointing with tar-ointment. In one very severe case I prescribed subcutaneous injections of osmic acid in the neighbourhood of the anus, which was highly successful; in another the result was less marked but still quite evident. One of my patients always felt comfortable for a few hours after he had violently pinched and squeezed the parts affected, but eczema, and the scars made by these manipulations ultimately made them impossible. Klein recommends cauterisation (*Ther. d. Geg.*, 1905). I have found hydrotherapy to be the most effective method of treating nervous urticaria. Washing with a weak carbolic or menthol solution has an alleviating effect. Rubbing with menthol ointment may be soothing, and euguform is recommended. The internal use of atropin, calcium chloride, phosphate of soda (60-75 grs. several times a day according to Wolff), etc., has been recommended. See further details in Joseph (*A. f. Kind.*, Bd. xxxviii.). The most important point is the general treatment and especially psychotherapy.

One final word as to the treatment of neurasthenia: Do not overdo the treatment. In recent cases, in which the exciting cause can be removed, it is better to abstain from any positive treatment. A powerful impulse will be given to the patient when he sees and hears that the physician, although in no way doubting the reality of his disease, leaves his recovery to the vis medicatrix naturæ. No rule can be laid down as to how far one should carry this principle. I have known cases of this kind in which the patient, after undergoing many courses of treatment in vain, renounced them all, returned to a rational mode of life, and thus regained his health.

Appendix

Morbid Conditions, which as a rule arise from Neurasthenia, Hysteria, or a Neuropathic of Psychopathic Diathesis, but which may occasionally be regarded as an Independent Disease

Conditions of Fear and Fixed Ideas

A.—Conditions of Fear, Phobias

We do not here propose to consider the conditions of fear which are produced by organic diseases of the brain, heart, etc.; but merely the anxiety which is of neuropathic and psychopathic origin. Fear is one of the most common symptoms of the neuroses. The feeling of anxiety is

sometimes vague and not awakened by any definite cause or dread; at other times it is produced by certain ideas and external causes. The feeling is described and localised in various ways. It is associated generally with the region of the heart, less often with the head, epigastrium, etc., but it may be quite indefinite. The patient feels as if his heart were standing still, as if he must fall, as if he had a stroke or had been sick, or as if something dreadful were about to happen. Another complains of sudden impairment of his consciousness of self, or a kind of breaking-up of his personality. He suddenly feels unable to think, *i.e.* he becomes aware of the inhibiting influence of anxiety upon the intellectual, and specially upon the associative processes. The expression of the face usually reveals the anxiety, which is accompanied by functional disturbances in the motor, secretory, sensory, and in particular the vaso-motor systems. The face becomes flushed or pale, and the rush of blood to the head may be associated with a vascular spasm in the distal parts of the limbs, *e.g.* coldness, pallor, and a feeling of rigidity and numbness. Perspiration breaks out, the secretion of saliva ceases, the tongue and lips are dry, the pulse and respiration quickened; there is a feeling of desire to pass urine and fæces, and polyuria, vomiting, and in particular diarrhœa may set in. Thomsen and Oppenheim [1] have found narrowing of the field of vision in cases of anxiety, and once during an attack of agoraphobia (see below). In the motor system the anxiety usually has the form of a feeling of inertia and want of strength, usually accompanied with great restlessness. Tremor is a frequent symptom. The anxiety is less often expressed by some involuntary motor action of violence.

Kornfeld,[2] in opposition to Janet, has found increase of the blood pressure, and he regards this exaggeration, or the increased vascular contraction combined with lessened activity of the glands, muscles, and the mental functions, as the actual cause of the condition, which is therefore one in which the central excitation is discharged only in one definite direction. But there may be no physical symptoms of any kind.

There are many indications that a congenital or acquired exaggeration of the excitability of the bulbar (specially of the vaso-motor) centres is an important factor in the origin and manifestations of anxiety. Whether these have no content, *i.e.* whether the anxiety awakened by certain ideas has its origin in them or is produced by unconscious mental processes, is unknown to us. But even when the feeling of fear is due to certain ideas and impressions, the cortical process cannot in itself alone be their cause; it must first create the feeling of discomfort by radiating to the bulbar centres. We therefore agree so far with James and Lange in ascribing to the physical processes produced by the bulbar centres a part in the production of the feeling of anxiety.

Brissaud, which whom Londe (*Rev. de Méd.*, 1902), Souques, and others agree, has insisted strongly upon the distinction between cortical fear (*anxiété*) and bulbar fear (*angoisse*). Hartenberg agrees with Morel in attributing the conditon to exhaustion of the sympathetic nervous system.

There is a class of cases in which the anxiety is only produced by the presence of certain external causes or in certain situations. Crossing an open space is the most frequent cause of anxiety. This *fear or anxiety of places*, Westphal's [3] *agoraphobia*, is a very common condition, which develops almost exclusively in *neuropathic* or *psychopathic* individuals, and chiefly in combination with neurasthenia. I have several times seen it alternate with an accessory spasm or with general tic (*q.v.*), or the two conditions appear in members of the same family. Chronic alcoholism

[1] *A. f. P.*, xv. [2] *Jahrb. f. P.*, xxii. [3] *A. f. P.*, iii. and vii.

is another cause of agoraphobia, and I have seen it in several instances follow injury to the head. I have no personal experience of a relation between this condition and diseases of the ear, which others have mentioned (Lannois, Fournier).

When the patient attempts to cross an empty square, in which there are no houses or people, a sense of anxiety comes over him ; he feels as if he could not move from the spot, as if he must fall, as if unending space stretched before him, etc. ; in slight cases he is able to conquer his dread and with a great effort to cross the open space. But on the other hand the anxiety, which he knows to be groundless and ridiculous, may so overcome him that he has to retrace his steps, to make his way across in the company of some other person, or to go round in the shelter of the houses.

When he has crossed the square, his anxiety vanishes. It does not usually affect him when he is near other people. One of my patients escaped from his sense of fear by leading a dog in a leash. There are cases in which crossing a carriage-way, or walking through streets which have not houses on both sides brings on the anxiety. Some of the patients suffering from agoraphobia only feel at ease when in a crowd ; others again become anxious when moving in a stream of people.

A great number of different *phobias* have been described, and the list could be extended at will, were it not undesirable to give new names to similar conditions. We speak, for instance, of a *claustrophobia* (fear of being alone in a narrow space), *anthropophobia* (fear of being with other people), *potamophobia* (fear of passing beside running water), *astrophobia* (fear of storms), *zoophobia* (fear of animals, *e.g.* mice, cats, snakes, etc.), *bacillophobia*, *aichmophobia* (fear of sharp, pointed objects), *kleptophobia*, *phagophobia* (fear of swallowing), *pantophobia* (fear of everything), *phobophobia* (fear of being afraid), etc. I have several times had occasion to treat persons with fear of railways and travelling, in whom the phobia was so intense that they had to stop in the middle of a necessary journey and were unable to continue it. The fear of a strange face has been described by Hartenberg, Bechterew, and Soukhanoff.[1] One of my patients made the extraordinary statement that she was afraid of looking at pictures and illustrations which showed many black figures, especially if she were alone. This dread seemed to date back to childhood. In others the anxiety is brought on by the sight of patterns on a wall paper, and so on. The disorder often makes itself felt in the exercise of the patient's calling. Thus, I have treated a clergyman who was afflicted by intense anxiety every time that he ascended his pulpit, so that he had for a time to abandon his calling. Bechterew[2] mentions the fear of giving the sacrament in priests ; Raymond and Janet speak of a professional abulia. The most common of all is *stage-fright*, *i.e.* the anxiety of actors, singers, etc., at the moment of appearing in public. There are some great artistes who never overcome it, and it may be so persistent and distressing that the stage as a profession has to be abandoned. The term of *fear of the situation* (topophobia) would comprise all these forms. Loewenfeld divides the phobias into locomotor, situation, and functional phobias.

An interesting case came under my care : one of my patients suffered from fear of storms, which was associated with physical symptoms, such as weakness, nausea, vomiting, etc. Her

[1] *Journ. de Psych.*, vi.

[2] *C. f. N.*, 1903.

little son of eight years old had no feeling of fear in a storm, but he also suffered from the same accessory symptoms.

In a few rare cases fear is the only morbid symptom, so that some writers (Hecker, Freud,[1] Hartenberg [2]) regard the " fear-neurosis " as an independent form of disease. As a rule it is associated with neurasthenic or hysterical symptoms.

I by no means agree with Freud's view that this symptom is caused by abnormalities of the sexual life in which absence of satisfaction plays an important part (coitus interruptus, frustrated excitement, etc.), although these may be a factor of great importance (see following section).

Nor do these conditions of anxiety appear only in people with a congenitally timid disposition. I have treated intrepid men and celebrated soldiers for this disorder.

The *prognosis* of agoraphobia and allied conditions of fear is not very hopeful as to recovery; the trouble is very *persistent*, but it tends to lessen in intensity as the patient learns to control his anxiety. There may also be long remissions. Complete and permanent recovery takes place only in a few cases. I have satisfied myself that this is so in a few cases in which the patient, who had previously suffered from agoraphobia, came to be examined on account of another disease. I know quite a number of such cases.

Mercier (*Lancet*, 1906) has published an interesting case of complete and permanent recovery from agoraphobia due to a severe mental shock (elopement of a daughter). I have sometimes succeeded in curing a condition of anxiety of some years' standing by simple encouragement. In one case a single consultation was sufficient. I learned from the husband of my patient, who came himself some years later to me for advice, that the one visit, or rather my definite assurance of her power to recover, had cured his wife of the trouble which had hitherto been so persistent.

Some writers include agoraphobia among the fixed ideas, to which it is usually related (see following section), and the term phobia (*e.g.* nosophobia, aichmophobia, misophobia, phobie du regard, fear of pregnancy, etc.) is undoubtedly applicable to conditions which belong entirely to the category of fixed ideas.

Treatment is practically the same as that of neurasthenia. As regards the special symptoms, the patient should at first for a considerable time avoid the occasions which produce the attacks of fear, but later, when the signs of exhaustion have disappeared, he should systematically seek by consistent and regular exercises to accustom himself to the situation. Hartenberg has advised the same line of treatment. *Psychotherapy* is of pre-eminent service. In agoraphobia, for instance, it would be well for the physician himself at first to accompany the patient across the empty square, and later to wait for him at the point on the other side at which he wishes to arrive. I have in this way induced complete recovery in some cases and great improvement in others.

In cases of anxiety from other causes, hot hand-baths, washing the head in cold water, etc., may be tried. Ziehen recommends passive movements and breathing exercises. Nerve tonics are often useful. It may eventually be necessary to give narcotics (opium, codein, dionin, hyoscin, etc.), or chloral in small doses (1·0 : 5·0, ten to twenty drops). " Nasal treatment " (removal of polypi or swellings) may do good in some cases (Hecker, etc.).

[1] *N. C.*, 1895. [2] *Rev. de Méd.*, 1901, and *Arch. de Neurol.*, 1903; *Rev. de Méd.*, 1904.

B. Imperative Ideas, Fixed Ideas (Westphal)

[Zweifelsucht, Folie du doute (Le Grand du Saulle, Falret), Délire émotif (Morel), Grübelsucht (Griesinger), Psychasthenia (Janet, Raymond)

The *history* of these affections is of comparatively recent date, although cases of the kind had already been reported by Esquirol, Baillarger, Morel, and others. The term "Zwangsvorstellung" (fixed idea) was first used by Krafft-Ebing, who did not, however, have a precise conception of its meaning. Griesinger went further in this respect. But the honour of clearly recognising and describing the nature of the disease and establishing its nosological independence undoubtedly belongs to C. Westphal (*B. k. W.*, 1877). The work of Falret, Le Grand du Saulle, Magnan, Charcot, Janet, Raymond, Freud, Thomsen, and others has greatly contributed to its investigation and analysis. We should also refer to the contributions of Kaan, Mercklin, Höstermann, Störring, Angell, Pitres-Régis, Oppenheim, Brissaud, Londe, Séglas, Mendel, Arnaud, Hascovec, Sollier, Soukhanoff, Friedenreich, etc. Loewenfeld has published a monograph ("Die psychischen Zwangserscheinungen," Wiesbaden, 1904) which includes a careful review of the literature—my contributions alone have escaped his notice. Later works on this subject are those of Raymond-Janet (*Nouv. Icon.*, xvii.), Pitres-Régis, "Les Obsess. et les Impuls.," Paris, 1902; Bonhöffer (*D. m. W.*, 1904); Crocq (*Journ. de Neurol.*, 1905); Fauser (*C. f. N.*, 1905); Bumke (*N. C.*, 1905); Pfersdorf (*M f. P.*, xv.); Friedmann (*M. f. P.*, xxi.); Warda (*A. f. P.*, xxxix.); Jung (*Journ. f. P.* vii.); Bumke ("Was sind Zwangsvorgänge?" Halle, 1906), and Raymond ("Névroses et Psychoses," 1907). See also Oppenheim, "Die ersten Zeichen der Nerv. d. Kindesalters," etc.

Ideas, which are not produced by association, but start up independently and intrude into the thoughts so forcibly that they cannot be banished, although they are recognised by the patient himself as being foreign and strange to his mental personality, are termed imperative ideas.

This is usually a symptom of a neurosis. Even when it seems to be an independent disease, we can discover the existence of a *neuropathic or psychopathic disposition*. The grave forms of the disturbance, indeed, always have this basis.[1] I have often been able to discover physical stigmata of degeneration in persons suffering from imperative ideas. Charcot and Magnan go too far, however, in thinking that this affection is always a stigma of degeneration or even of hereditary insanity. Janet and Raymond apply the term psychasthenia to these conditions.

Their development is favoured by conditions of exhaustion and sleeplessness. In some of my cases they were brought on by news of some misfortune (*e.g.* hearing that a neighbour had hanged or shot himself), in others by treatment at a watering-place (Karlsbad). As a rule, the onset is sudden. The imperative thought, which the patient recognises as being in itself strange and peculiar, comes to him without any reason. He tries to throw it off, but the more he strives to get rid of it, the more firmly does it cling to him, breaking violently into his train of thought and seeking to dominate it entirely. The import of the ideas is very variable. They are often thoughts which come at times to every one, but are quickly banished

[1] I have treated three members of one family for imperative ideas, and was able to ascertain the following family history: The father was in his youth an epileptic, and later an eccentric character. A sister of the mother suffered sometimes from hemeralopia, sometimes from rumination Of the six children, one daughter was afflicted with hypochondriacal insanity, one son with epilepsy and mental equivalents, another with neuralgia, and the three others were under my care for imperative ideas—one for morbid doubt, a son for perverse sexual sensations with anthropophobia, and his brother for the fixed idea that he must wound or kill some one. In every case the intelligence was normal.

because of their illogical character, or ideas such as occupy the normal mind at times with great intensity but without obtaining the mastery and becoming painful and troublesome by their persistency. Among those of the first category are the impulses of the patient to throw himself into a river or down a precipice, or the thought of wounding himself suggested by the sight of a sharp instrument, etc. Such ideas occur to healthy people, but they are very transient and can at once be suppressed. If they constantly return, refuse to be banished, and are associated with intense feelings of uneasiness, they represent fixed ideas. At other times they take the form of *questions* with regard to God, to the world, to the destiny of the human race, questions which certainly occupy every thinker and are morbid only in that they constantly obtrude into his thoughts and cannot be dislodged. This obsession of doubt [1] becomes peculiarly distressing when almost every idea assumes the form of a question, when every idea conveyed by the senses, every action, suggests the query: "What is this?" "Why am I doing this?" "Why do I do it in this way and not in some other?" "Why does that object stand there?" etc. These questions are quite unreasonable and the patient recognises their absurdity or folly, but is unable to suppress them. Or the imperative ideas may in themselves be quite absurd and show no relation to the patient's normal thought-processes. Thus one of my patients was troubled by the idea that he was carrying the head of his dead father below his arm, that his skin was a mouse-trap, etc. There are other cases in which the patient worries himself by thinking of *names*. I have treated a woman who was at great pains to find the name for every object and could not rest until she had written them down. She had sacks full of bits of paper covered with names. In other cases there is a kind of impulsive orientation and analysis. The patient is forced to recollect exactly what he was thinking and doing at a certain time, what objects he saw in passing through a room, in what order they passed before his eyes, etc. etc.

Many of the people afflicted with this impulse to question and to orient themselves are congenitally of an anxious, pedantic, and scrupulous disposition.

The occurrence of sacrilegious thoughts, ideas, and impulses during prayer is very painful. The patient desires to follow his devotions, and an oath or obscene word comes into his mind and refuses to be banished. The mania may have reference to *figures*: the patient is compelled to count the windows of a house, the steps in a flight of stairs, etc.

There is also a pathological impulse to fall in love (Laurent); this hypererosia (Jastrowitz [2]) may have its origin in a mental disorder, but it may be a stigma of degeneration.

Frequently the imperative idea—which is then to a certain extent physiological—refers to some action which has not been correctly performed, a letter unaddressed, a cupboard not shut, etc. I have had under my care several lawyers and doctors who were excessively worried by the fixed idea of having made an error in filling up a document, or writing a prescription. The fixed idea of being morally lost on account of some action is not an uncommon one. I have treated a very intelligent lawyer who

[1] Loewenfeld distinguishes these forms of imperative doubts, etc., in which there is a tendency to form ideas all of the same character, or related to each other by their contents, from the isolated fixed ideas; he calls the former "associative imperative tendencies or doubts," and includes among them the mania of doubt, the metaphysical mania, imperative scrupulosity, etc.

[2] *D. m. W.*, 1903.

had provided his windows with shutters ; subsequently he developed the fixed idea that in doing so he had committed an act of cowardice. He was terribly troubled by this thought of moral inferiority, and consulted not only doctors, but philosophers, clergymen, etc. When he asked my advice, he had already suffered from this trouble with intermissions for twenty-five years. The idea of having hurt other people by omitting to perform some action (*paralipophobia* of Ziehen) is closely allied to this form. The thought of becoming poor may also take the form of a fixed idea.

In some very severe cases the thoughts and actions of the patient are dominated by the fear of *soiling* himself. He knows well that certain objects are not dirty, but in spite of that knowledge the fear of dirt (misophobia) lays a spell upon him ; he avoids touching latches, money, and many other things (*délire du toucher*), or is very often, one might say, continuously compelled to wash his hands. For example, a woman had seen some one run over ; immediately afterwards she heard that the intestines had been seen protruding from a wound in the abdomen, and she was at once obsessed by the thought that she and all the objects around her were soiled thereby. She was quite conscious of the absurdity of the idea, but could not get rid of it for years. Sometimes it is the idea of taking the life of another, especially of the nearest relatives, which absorbs the thoughts and becomes very distressing. One of my patients could not cross the street because he imagined that he might hurt some one with his stick or umbrella. In others the idea that one has become deformed, that one's clothes do not fit, may assume a very troublesome character.

There is another condition in which *ideas* and *opposing ideas* may be *imperatively* felt. A young and highly intelligent woman reproached herself greatly because she imagined that she was wishing some evil to befall others, her friends in particular. As soon as she thought of any particular object (ornament, dress, painting, etc.) which she knew to be in the possession of some one else, the thought arose, "*You* would like to have that" ; this was followed by the other thought, " on this account *you* are wishing some harm or even death to X." She then felt compelled to compose sentences intended to act to some extent as a protection against these imaginations, something as follows : " I do not wish any ill to X." A doctor who wished to marry, was troubled by the fear of impotence, but found that this was groundless. One day the thought suddenly shot through his mind that his sexual organs were shrivelled and atrophied. He could no longer throw off his fear, although he recognised its groundlessness. He then tried to paralyse the primary idea by counter-ideas, as he expressed it, but these in their turn became imperative thoughts, and the idea that his sexual organs were well developed, which now dominated his mind, persisted, without bringing him any relief.

These imperative thoughts are not infrequently translated into irresistible movements and *irresistible acts*, the carrying out of which at first brings a certain relief and calmness. But these impulsive actions are usually not executed, if they are such as to compromise the sufferer and bring him into conflict with the law. This fact is noted by Janet and Raymond, who have had wide experience. There are exceptions to this rule, however. I have treated a student who was under the control of the idea that he must spring upon some one else and wound him. He

had always been able to master this thought, until once during a lecture, when the fear connected with this idea overwhelmed him to such an extent that he flung an inkstand at the blackboard.

Loewenfeld classifies imperative movements and acts as follows: 1. those which are intended to test, improve, or supplement the results of previous acts; 2. protective or remedial measures, *e.g.* the mania of cleanliness caused by fear of bacilli, etc.; 3. those intended to improve the conditions, which may be impulses of an indifferent nature (such as the mania for orderliness and cleanliness) or of a criminal or suicidal character.

Imperative ideas may have an inhibiting influence upon the normal will power, preventing the patient from executing certain acts, movements, etc., and reducing him for a time to a condition of complete perplexity.

Janet and Raymond speak of the mental stigmata of the psychasthenic, amongst which they include the feeling of incompleteness, the inhibition, abulia, depersonalisation, etc.

The *conditions of anxiety* described in the foregoing section are closely allied to *imperative ideas* and cannot usually be sharply distingushed from them, but they exhibit certain peculiarities which make it desirable to regard them as a special type or variety. For one thing, the intellectual processes take little share in these conditions, which are characterised by the *rapid translation of the idea or the sensory impression into a feeling of anxiety*. There is usually in addition an abnormal excitability of the vasomotor and other centres which are only indirectly or not at all under voluntary control. The agoraphobe is usually free from every morbid idea when in his own room or walking with a companion. When he tries to cross an empty square, morbid fears (such as the thought that he will fall down or have a fit, etc.) often overpower him, but in other cases the condition of anxiety comes on without any cause or any definite conscious idea; indeed, the visual memory image seems to be so closely associated with the pathological emotion that it is in itself quite sufficient to bring on the attack of anxiety. The fear may in the first instance have been due to some definite occurrence and to his consciousness of helplessness, as the presence of a companion can avert the attack of fear. The same criteria distinguish from pure imperative ideas the cases in which the desire to pass urine or fæces makes itself distressingly felt whenever the patient is in a room with many people, *e.g.* at a theatre, concert, social party, or table d'hôte; this is seldom accompanied by actual incontinence (urophobia, closet-mania, etc.). I have discussed this form, which has hitherto received little attention, in a Russian journal (*Medicinskoe Obosrenje*, 1901). The trouble generally arises from the desire having once occurred under circumstances which made it impossible to satisfy it immediately. The remembrance of this situation always evokes the idea under similar external conditions, and thus gives rise to the imperative desire, and possibly to a spasm of the sphincter or a spasmodic contraction of the detrusor. The impulse to vomit and actual vomiting may arise under similar conditions. A lady who had once been overcome with sickness at a social gathering, was afflicted with fear of vomiting (Oppenheim, Bechterew) whenever she began to dress for a party. I have even seen a hereditary family form of this trouble. The patient suffers, he assures us, not from the idea or imagination itself, but from the condition of anxiety to which it gives rise. The *bégaiement*

urinaire (Harnstottern, bladder-stammering) described by Paget, Guyon, Janet,[1] Raymond,[2] etc., which consists in inability to pass urine in the presence of others, may develop into painful retention; here also the inhibition of function is due to the imagination.

This is the case also with regard to "morbid blushing," "erythrophobia," or "ereuthophobia," in so far, at least, as concerns not the vasomotor act, but the uncomfortable sensation which is associated with the idea or fear of blushing (Casper, Eulenburg, Pitres-Régis,[3] Bechterew,[4] Friedländer, Hascovec, Vaschide-Marchand, Claparède,[5] etc.). Here the predominant part played by exaggerated vasomotor excitability is very evident, but it is clear that this factor does not alone constitute the morbid condition. We may with Hartenberg[6] and others distinguish between the morbid exaggeration of the tendency to blushing (ereuthopathy) and the form associated with or based upon conditions of anxiety and imperative ideas (ereuthophobia), but one condition may develop from the other. It is only if we assume that the anxiety is a feeling of discomfort produced by the vasomotor processes that we can regard the whole condition as one of abnormal excitability of the vasomotor system and thus distinctly different from the imperative idea (see above). The fact that abnormal excitability of the vasomotor centres is an essential element in the production of the symptoms is shown by the other troubles of which the patient complains, one of the most common being coldness of the feet and hands. In one of our cases the influence excited by the imagination upon the vasomotor system reached a very high point: A young, healthy woman, who could stand the sight of blood quite well, was, especially in the presence of other people (at a theatre, concert, in the street), possessed by the idea that she saw a man bleeding from the nose. This imagination immediately produced a state of violent anxiety, which was so excessive that during some of the attacks she fell down unconscious and involuntarily passed urine and fæces.

Many writers have discussed the classification, delimitation, and *psychological analysis* of imperative ideas, but we can only consider a few of their views. Westphal's theory, that the primary and essential element of imperative ideas is a purely intellectual (non-emotional) process, has been much opposed, and it certainly cannot be maintained in its full scope, but it does apply, in the opinion of Thomsen and myself, to the typical forms of imperative ideas, metaphysical mania, etc.

Most writers lay chief stress upon the *congenital disposition*, and we think there is no doubt that the typical, severe forms, to which Westphal's definition relates, develop on this basis. In such cases the disease may acquire such independence that we are justified in speaking of it as an imperative affection of the imagination or an imperative neurosis. On the other hand phobias and imperative ideas may undoubtedly be due to and associated with neurasthenia (and hysteria) and may be equivalent to the other symptoms of these neuroses. Janet distinguishes the affection from these neuroses, regarding it as a *psychasthenia* which is allied to epilepsy. See also Ziehen's remarks upon *psychopathic constitutions* (*Charité-Annalen*, xxix.).

If imperative ideas were always congenital in their nature one could always rest satisfied with the explanation that thoughts of this kind were due to congenital disposition. But their sudden and often causeless occurrence in subjects who have hitherto been healthy or merely neurasthenic, has compelled one to look for another explanation. Friedmann lays stress upon the fact that the thought or sequence of thoughts are left unfinished; for this reason worry, fear, expectation, and doubt form the object of the imperative idea. This does not greatly help to explain the pathogenesis. Loewenfeld thinks that the fundamental characteristic of the

[1] "Les troubles psychopath. de la miction," *Thèse de Paris*, 1890.
[2] "Leçons," 1898. [3] *Arch. de Neurol.*, 1897. [4] *Obosrenje*, 1895; *N. C.*, 1897.
[5] *Arch. de Psychol. de la Suisse Rom.*, 1902. [6] *Rev. de Méd.*, 1902.

imperative ideas is the impossibility of suppressing or changing them; that is merely another way of stating established facts. But he has endeavoured to discover the causes of this characteristic, and thinks it is due to the emotional origin of the ideas and to exhaustion of the nervous system. The intensity of the sense of uneasiness, accompanied or awakened by the idea, gives it a lasting character and makes it more easily reproduced, the association of an idea with a sense of pain, once produced, giving rise to the conditions for an associative blending of the two. Exhaustion of the nervous system, the result of the neurasthenic disposition, and a condition of exhaustion accidentally present at the first onset of the imperative idea, by weakening the will, diminishes the power of changing the idea, just as emotional excitement reduces the associative faculty, etc. He describes two main classes: imperative ideas of the *intellectual* and of the *emotional* spheres. Among the latter he includes conditions of anxiety and phobias.

Freud's ingenious analysis is exceedingly original. He states that if a predisposed person, by the suppression of some intolerable idea, usually connected with his sexual life, refuses to give it emotional expression, this emotion must remain shut up in his mind. The idea, thus weakened, persists, apart from all its associations, in his consciousness, and the emotion which has been detached from it becomes connected with other, not intolerable ideas, which thus, on account of the false association, become imperative ideas. The mental processes which connect the effort of will, directed to the suppression of the painful idea and the onset of the imperative idea, are unconscious in their action. He has subsequently defined imperative ideas as being *altered self-reproaches constantly recurring in spite of their repression*, which always (or mostly) relate to some sexual, pleasurable action of childhood. Warda (*A. f. P.*, xxxix.), Jung, and others have accepted Freud's theory, but it is by no means applicable to every case. Janet thinks the essential element in the development of fixed ideas is mental deficiency and weakness of will-power; Arnaud thinks the latter is the most important cause.

Taking everything into consideration, we may say that typical imperative ideas (mania of doubt, metaphysical mania, etc.) are due to a special congenital disposition of the neuropathic-psychopathic nervous system. The first symptoms are as a rule manifested early; at least the patient from his birth shows a peculiar, pedantic nature, dominated by scruples. This disposition, however, exists to a minor degree in every neurasthenic person, and some cause, such as some violent emotion or its coincidence with a condition of exhaustion, is required to produce the special affection. This is particularly true as regards the genesis of simple phobias (the situation phobia). Freud's explanation applies to a large number of these cases.

With regard to the diagnosis, confusion between imperative ideas and hallucinations must be carefully avoided. The mental personality of the patient is in sharp opposition to his imperative ideas; he recognises that they are something alien and intrusive, and is *quite aware of his morbid condition*. Hallucinations, on the contrary, have become a part of the patient's mental content; he does not simply think he is pursued; he is convinced of it. Sensory hallucinations are seldom combined with imperative ideas (cases of Kelp, Ballet, Loewenfeld), and it is very doubtful, as Mendel and Jolly state, whether such cases should be classed with imperative ideas. A patient suffering from imperative ideas is not mentally unsound, and should not be regarded or treated as if he were so. A great number of such patients are capable of any mental work, and learn to control themselves so well that the condition would never be noticed, unless the patient himself mentioned it. In other cases, of course, the imperative ideas intrude so violently and dominate the thoughts so completely that the whole mental life is stifled, the patient's insight is, for a time at least, suspended, and his responsibility becomes doubtful. It must also be admitted that imperative ideas may be associated with true psychoses, especially with paranoia and with severe hypochondria and

melancholia (Tuczek, Mercklin, Heilbronner, Juliusburger), but this symptomatic form should be distinguished from the true one. I have seen several cases in which periodic insanity was in each attack combined with and apparently based upon the same imperative idea. In some of these the idea related to sexual aberrations.

Men and women in middle life are almost equally liable to this trouble, but it may occur in youth and even in childhood (especially in its typical forms of mania of doubt, metaphysical mania, fear of contamination, etc.). I have indeed seen very severe cases of this kind in children, and it may be exceedingly difficult to recognise the condition under these circumstances. The affection is sometimes combined with general tic or accessory spasm, or it occurs in families in which the other members suffer from these neuroses. The relation between the tics (see chapter upon this subject) and imperative ideas is a very intimate one. Janet classes them all together as "*agitations motrices systematisées.*"

The *prognosis* of imperative ideas is grave, or at least doubtful. The phobias of situation have on the whole much the most favourable outlook. I have also known a number of cases in which the patient got entirely rid of his fixed ideas, or learned to pay so little attention to them that the condition became quite bearable. In other cases there were remissions and exacerbations, which did not, however, give the disease a serious character. I have often learned from patients whom I have been treating for other nervous disorders that they have at some previous time suffered from imperative ideas. In one lady they had developed after a course of treatment at Karlsbad; she had afterwards been free from them for ten years, but they again returned when she was in a state of great exhaustion.

One young lady of twenty-six, who consulted me for stuttering and other nervous symptoms, told me that she, from the age of eleven to thirteen, had suffered from misophobia. Another woman of sixty, who was under treatment for the mania of blasphemy, had forty and again twenty years before been subject to the same impulses, and since then had a period of twenty years' relief from them. One man of fifty-four, who consulted me on account of insomnia, had suffered from his fifth to his eighteenth year from the *folie du doute avec délire du toucher*, which had since then entirely disappeared. One of my patients had for years the mania of washing; she subsequently developed a typical Raynaud's disease, and it seemed as if the constant effect of the cold water had helped to bring on this disease. In a number of other severe cases, in which the disease had for years proved intractable, I was able by systematic and long-continued treatment to effect more or less complete recovery. The condition very rarely develops into a *psychosis*; Janet thinks this occurs in twenty-three out of three hundred cases, but from personal experience I should think this number too high. Suicide on account of imperative ideas is also a rare occurrence. Janet has never observed it, and I do not recollect any case in which the typical uncomplicated condition has led to suicide.

The typical forms of misophobia apparently represent the extreme height of the affection and make the deepest impression upon the patient's mind.

Treatment is practically that employed in neurasthenia (see preceding section); mental treatment is the most important factor. The definite assurance of the physician that no mental disease is present or to be feared

is often in itself of great service. Suggestion must enter into every kind of treatment. I have found hypnotism useful in some obstinate cases,[1] but in a great number of others it has completely failed. By entering fully into the mental life of the patient one is best able to understand how the pathological associations have arisen and to find the indications for the line of treatment. Every patient bears himself differently in his confidential talk with the physician; most of them long for this opportunity and find great relief from their talk, but it seldom happens, as in a patient of Claparède's who had erythrophobia, that this conversation is in itself sufficient to effect a cure. In other cases every reference to the subject intensifies the trouble. The way in which the patient unburdens his mind during his consultation with the physician is very characteristic; his facial expression often reveals the fact that he is distressed about something which he is reluctant to confess.

Ample occupation is a very important point in the treatment. Physical work, such as farming, gardening, mechanical labour, rowing, sailing, motoring, etc., and such occupations as botanising, making microscopical drawings, painting, drawing, modelling, chess-playing, etc., may be recommended. For ladies, into whose occupations it is particularly difficult to introduce variety, translating books into the Braille type for the use of the blind will be found an engrossing task. Travelling, which brings with it a great variety of fresh interests and beauties, may be very helpful.

Freud's *psychoanalytic method* has been developed out of Breuer's eliminative method (see p. 1104). It turns to account the "expanding of the consciousness" which takes place in the hypnotic state, as the patient when in this condition reverts to the state of mind in which he was when the symptom occurred for the first time. Both writers explain the therapeutic effect of their method by the fact that it leads to the discharge of the pent-up emotion which has become attached to the suppressed mental processes ("Abreaction"). Freud, recognising the deficiencies of this method, resolved to abandon hypnotism and to confine himself to conversations with his patient, during which he sits behind him and is therefore out of his sight. The talk should be quite unrestrained and should reveal every whim of the patient's mind, *i.e.* he should give expression to the involuntary thoughts which occasion him distress and are therefore suppressed under ordinary circumstances, and which tend to break into the connected account of his case which he is trying to give. He should say everything as it comes into his mind, even although it seems to him unimportant and foolish. In this way the amnesias caused by the repression of his thoughts become evident. The vagaries of thought which under other circumstances he conceals by every kind of pretext, are regarded as offshoots of the suppressed mental processes, as misrepresentations of these due to the resistance opposed to their reproduction. Freud thus attained to a *science of interpretation* and made use not only of the ideas of the patient, but also of his dreams, which opened the most direct access to knowledge of the unconscious processes. He believes that he obtains in this way an insight into the play of the psychical forces and discovers the suppressed idea which he regards as the root of the compulsive thoughts, etc. etc. He thinks that the aim of treatment should be the abolition of the amnesia.

I have here described this method, which requires for its accomplishment six months to three years, because an intelligent physician will always find it an interesting task to discover the root of such an intractable trouble. But apart from the fact that it depends upon theories which are certainly by no means applicable to every case, I regard this kind of procedure as dangerous to many of the patients, and I base this

[1] I had given this treatment fifteen years previously to a patient suffering from *folie du doute*. She returned to consult me quite recently on account of other symptoms, and assured me that she had been absolutely free from her fixed ideas during the whole intervening period.

view, not merely upon theoretical considerations, but upon a case which came under my observation after having been treated in this way for three years.

Out of the many attempts which I have myself made to discover a rational way in which to treat these fixed ideas, I should like to describe only the following :—

I exercise the patient in voluntary interruption of thoughts which usually follow each other in definite order in the memory; for example, I prompt him to repeat in his mind the days, months, figures, familiar verses, etc., and then to break off at a given sign; or I say the first words and he tries to become accustomed not to continue thinking them on to the end, but to fix his attention upon something else. This method has only been successful in a few cases.

Astasia-Abasia

The disturbance of function to which this name is given has been known for a long time, but has been specially studied of late by Blocq,[1] Charcot, Pitres, Binswanger, Ziehen, Thyssen, Delarue, Strohmayer,[2] Trömner,[3] etc. It is not an independent disease, but is rather a symptom or group of symptoms arising from *hysteria*, or less commonly from neurasthenia, hypochondria, etc., and most often excited by some *outburst of emotion* or by some *injury*. It has also been observed after an *infective disease* (typhoid) and CO-poisoning.

The condition is one in which the power of standing and walking is greatly impaired or quite abolished, although no disorder of motility, sensibility, co-ordination, etc., nor any mechanical hindrance to the movements of the legs can be discovered when the patient is in the recumbent position. When in bed he can move the legs freely and with normal power and co-ordination, but when he tries to stand and walk he collapses or drags himself along with difficulty. If walking is merely difficult (*dysbasia*), the trouble may be simply due to an insufficient output of muscular force, to inco-ordination which is only present during this action, or to involuntary movements (of choreiform nature or tremor). In the majority of cases the inability to walk is absolute. The patient can move himself on all-fours. The power to swim and perform other locomotor actions may be retained. The patient can sometimes walk backwards.

Abasia is usually a symptom of hysteria and is to be compared with aphonia and similar functional disturbances. It often seems to be due to a loss of the memory pictures for the act of walking.

I have also observed some cases which indicate that the trouble may belong to the class of *occupation-neuroses* (*q.v.*), and may be analogous to writers' cramp. Here as there, there is disturbance of the co-ordination of the muscles for some definite action, although they act in the normal way for every other purpose; here also the motor power, which is otherwise intact, is impaired in carrying out the movements necessary for walking and standing. The disturbance may also be due to abnormal contraction (the spastic form—basospasm—but this should not be confused with the idea conveyed by the word spastic in its strict sense), to weakness (paralytic form), to tremor, and finally to the fact that abnormal innervation of the muscles in walking and standing brings on pain.

In cases of the latter kind, walking is not usually absolutely impos-

[1] *Arch. de Neurol.*, 1888. [2] *M. f. P.*, xii.; contains bibliography. [3] *N. C.*, 1907.

sible; the inability appears after the patient has taken a few steps, or he merely feels great difficulty in walking. There are no hysterical symptoms, but (as in writers' cramp) some of my patients were neurasthenics. In one case *over-exertion* of the muscles of the leg, and in another a painful condition in the foot had preceded its onset. Care must be taken not to confuse the condition with intermittent claudication (see p. 586), although I have described a form of this disorder caused by functional disturbances.

Strohmayer has noted that functional disturbances in the visual apparatus, such as weakness of accommodation, may help to produce this syndrome. Bruns speaks of a "stuttering of the legs." There are also cases in which a feeling of anxiety befalls the patient when he tries to walk, or the imperative idea that he is unable to do so causes the inhibition (stasobasophobia of Debove, Mingazzini, Ballet, Duprè-Delorme, Sainton,[1] and others). These forms are allied to the conditions discussed in the chapter on agoraphobia. Intense self-attention and introspection may be a disturbing factor in the automatic action (Oppenheim,[2] Pick[3]).

This condition is naturally most common in young people, and to some extent in women. *Cerebellar ataxia* is a disturbance of gait to which the above definition might apply, but it is so definitely characterised that there can be no difficulty in diagnosing it. Bonnier speaks of an astasia-abasia of labyrinthine localisation. Compare also the observations of Petrén given on p. 821.

The *prognosis* is favourable, but the trouble may be very persistent. As regards treatment, consult the chapter on hysteria and neurasthenia (including agoraphobia). Psychotherapy and methodical gymnastics are particularly helpful. Recovery can often be induced in children by simple suggestion.

On the other hand it must be recognised that disturbances of gait caused by organic diseases of the central nervous system may be intensified by the addition of mental factors, and also that basophobia may be superadded to an organic affection.

The "akathisia" (inability to sit down) described by Haskovec[4] is a symptom allied to abasia, but is usually a form of phobia (Raymond-Janet[5]).

Akinesia algera (Möbius)

Under the name of "akinesia algera" (ἄλγηρος,, painful), Möbius[6] has defined the following symptom: voluntary absence of movement on account of the pain which it produces, and for which no cause can be assigned.

Cases of this kind have been described by Erb,[7] Longard, Mingazzini, Bechterew, Oppenheim,[8] Fiorentini, Ingelrans,[9] Scheikewitz,[10] and others.

This is not an independent disease, but is *a symptom* or *syndrome* arising from neurasthenia, hypochondria, hysteria, and mental degeneration. At first pain is only caused by certain, and especially

[1] *Gaz. méd. des hôp.*, 1903.
[2] "Psychoth. Briefe," Berlin, 1906; Edin., 1907.
[3] *W. kl. R.*, 1907.
[4] *Arch. bohém.*, 1902; *Nouv. Icon.*, xvi.
[5] *Nouv. Icon.*, xv.
[6] *Z. f. N.*, i. and ii.
[7] *Z. f. N.*, iii., v. and vi.
[8] This Textbook, and "Zur Prognose und Therapie der schweren Neurosen," Halle, 1902.
[9] *Gaz. des hôp.*, 1905.
[10] *N. C.*, 1907.

by forced movements. Gradually every movement becomes painful; the pain lasts longer than the movement, and extends to other parts of the body which have not been moved, until finally there is complete inability to move, which is due not to paralysis but to pain. The patient has the appearance of being completely paralysed.

The pain is obviously a psychalgia—hallucination of pain (Möbius, Erb)—and is not, as Bechterew thinks, of a physical nature. This symptom, which is the chief one, is associated with other signs of *neurasthenia* or *hypochondria*. The tendency to grave *psychoses* is sometimes marked.

In a case of this kind under my care the attacks of pain were accompanied by very rapid respiration and pulse. Passive movements also produced pain. In another case the symptoms were limited to the right side of the body and were associated with hemianæsthesia. Slight degrees of this trouble are often present in the course of the traumatic neuroses.

In a similar way other functions may be impaired or completely inhibited by the pain which they evoke. There is a *persistent form of visual disorder* (dysopsia algera), caused by the fact that using the eyes, and especially fixing them on any point, gives rise to pain in the eyes and head. In several of my cases the pain was only brought on by looking at white objects (paper, linen, snow, etc.). The chief complaint of nervous persons sometimes is that any attempt to read causes a feeling of discomfort (pressure, or itching in the head, or even dyspeptic symptoms). In a case of Erb's, pain was specially evoked by *listening*, so that the patient could not bear a conversation, and was restricted to monologues. Indeed, the pain had compelled him to occupy a recumbent position for fourteen years. I have treated a lady who felt pain after every *meal*, and had consequently become extremely weak. Whilst this lasted the patient was comparatively well, but when she recovered to the slightest degree the pain re-appeared, and was accompanied by rapid action of the heart, vasomotor symptoms, polyuria, etc. There was, of course, no gastric disease.

The condition described by Neftel as "atremia" probably belongs to this class. In it the patient is confined to bed on account of the pain, anxiety, and disturbances of the general health which accompany standing, walking, sitting, etc.

The *prognosis* of all these conditions is grave, or not encouraging. The patient usually shows a very marked hereditary tendency to disease, and the *apraxia algera* is merely the culminating point of a long illness. Recovery is, however, not impossible. I have been able to cure one case in which the pain occurred whenever the eyes were used by prescribing blue glasses, galvanic treatment, and arsenic. In Erb's case and in the very severe case of *aphagia algera* already mentioned, the patients have reported themselves cured. Another patient of mine, who had been confined to bed for a year and a half and was regarded by several eminent physicians as incurable (on the assumption that the disease was an organic one), completely recovered her health under the regular treatment which I gave her for six months. This recovery took place eight years ago and has proved complete. The cure in such cases is never rapid or miraculous but is the result of prolonged, consistent training, which can usually only be carried out in a sanatorium. I have found systematic exercises, in which the demands made upon the patient are gradually increased, the

continuous bath, and subcutaneous injections of duboisin most helpful in such cases. (Consult, however, these and other observations in my paper: "Zur Prognose und Therapie der schweren Neurosen." Sammlung zwangloser Abhandlungen, Halle, 1902.)

Another case of this kind, the worst, indeed, which I have seen, has been under my care for a number of years, and the condition, which has persisted for ten years, has gradually improved to an extent that may be called incomplete recovery. Here each small advance had to be laboriously achieved, and the result was only attained by great consistency and patience.

As to further treatment, consult also the previous chapters.

The Traumatic Neuroses (Neuroses of Accident)

Injuries may affect the nervous system in various ways. The symptoms and clinical conditions caused by gross lesions of the brain and spinal cord and of the peripheral nerves have been discussed elsewhere.

Great interest is attached to those morbid conditions which arise from *concussion*, whether directly affecting the central nervous system or being conveyed to the brain by means of the sensory nerves. The symptoms are in many points identical with those of the neuroses and psychoses, especially with those of *hysteria*, *neurasthenia*, and *hypochondria*. The clinical condition is sometimes exactly similar to that of one of these neuroses, but is usually produced by a *combination of hysteriform and neurasthenic symptoms*, and these again are often associated with symptoms and groups of symptoms which extend beyond the limits of hysteria and neurasthenia, but are nevertheless due to functional disturbances (*reflex epilepsy*, *epilepsy*, *reflex neuroses*, *localised muscular spasms*, *psychoses*, etc.). The syndrome may therefore be a very complex one. The neuropathic and toxic diatheses (alcoholism, lead-poisoning, etc.) facilitate the development of these neuroses.

Our knowledge of these morbid conditions is of comparatively recent date. They had, it is true, been observed and described by earlier writers, but their explanation of them and the conception which they formed were incorrect, as they supposed the symptoms to arise from an organic disease of the spinal cord (a chronic meningomyelitis, Erichsen,[1] Riegler[2]), the brain, or the peripheral nervous system. I think that the two first cases of nerve-stretching, described by Billroth and Nussbaum in 1869 and 1878, must have been instances of traumatic neuroses.

Our present conception of the condition was first introduced by the investigations of Walton-Putnam,[3] Thomsen,[4] and Oppenheim, and subsequently the whole matter was worked up simultaneously by Charcot[5] (and his pupils) and by Oppenheim[6] (as well as by Strümpell,[7] Page,[8] and others). See also Schultze, Volkmann's "Samml. kl. Vortr.," *N. F.*, Nr. 14, 1890; Bruns, *N. C.*, 1889; Freud, Volkmann's "Samml.," *N. F.*, Nr. 51, 1892; Strümpell, Wernicke, "Ref. über traumat. Neurosen auf 12. Kongr. f. inn. Med.," Wiesbaden, 1893; Strümpell, "Über die Unters., Beurt.," etc., München, 1895; "Obergutachten der med. Fakult. d. Univ. Berlin," etc., *M. f. Unf.*, 1897; Bruns, "Unfallsneurosen," "Enzyklop. Jahrb.," viii., 1898; Sachs-

[1] "On Concussion of the Spine," etc., London, 1876; second edition, 1882.

[2] "Über die Folgen d. Verletz. auf Eisenb.," etc., Berlin, 1879.

[3] *Boston Med. and Surg. Journ.*, 1883 and 1884.

[4] *C. f. d. m. W.*, 1884; *A. f. P.*, xv.: also Oppenheim, *B. k. W.*, 1884, and *A. f. P.*, xvi., etc.

[5] "Nouveau Traité sur les Maladies des Nerfs"; German translation, 1886; also *Prog. méd.*, 1885, 1886, and 1887; "Leçons du Mardi," 1888-89.

[6] "Die traumat. Neurosen," first edition, Berlin, 1888; second edition, 1892; *B. k. W.*, 1888.

[7] *Berl. Klinik*, 1888.

[8] "Injuries of the Spine," etc., second edition, London 1885; German translation, 1892.

Freund," Die Erkrank. d. Nerv. nach Unfällen," Berlin, 1899; Bruns, " Die traumat. Neurosen, Unfallsneurosen," Nothnagel's " Handbuch," xii., 1901, containing bibliography; Schuster, " Deutsche Klinik," etc., 1905; Bailey, " Diseases of the Nervous System resulting from Accident," London, 1906.

In his clinical lectures of 1887-88, Charcot says: " These cases were until three years ago unknown to me and to others." This is an important fact, as it contradicts the view that the present investigation in this field is merely a revival of old and well-known views.

Mental shock—fear and excitement—plays an important part in the *etiology* of this morbid condition; indeed, it is the sole cause of the trouble in some instances. Accidents in which a physical trauma is associated with a mental shock, as in a *railway accident*, are specially liable to produce this neurosis. These have indeed been the main source of our knowledge of traumatic neuroses. But any injury, even although it involve merely some peripheral part of the body (hand, foot, etc.), may result in this condition. In such cases, however, the injury has usually been accompanied by severe concussion of the affected part or by some great mental shock. Thus, I have occasionally seen severe neuroses follow a heavy fall or blow on the finger-tips or prolonged crushing of the fingers. Some of the paralytic conditions following a *stroke of lightning* (kerauno-neurosis) should be regarded as traumatic neurosis. In such cases symptoms of the functional neurosis are often associated with signs of an organic nervous lesion; I have recently noted this in one particularly severe case. Traumatic neuroses have been often observed of late after an electrical shock (contact with electrical currents, falling of the conducting wires of electric railways, etc.). I have seen several such cases, and they have been specially studied by H. Strauss, Eulenburg,[1] and in particular by Hoch.[2]

The foregoing observations, which agree with those of Jessen (*M. m. W.*, 1902), Panas, and others, and specially the careful work of Jellinek [3] and Battelis,[4] indicate clearly that the danger to life and damage to the nervous system depends not merely upon the tension, but also upon the duration, nature, and site of the stimulus, and the condition or power of resistance of the skin at the point at which the current enters the body, the intensity of the current, etc. Thus under certain circumstances death occurs at 155, or even at 95 volts, whilst at other times 2000-3000 volts can be borne. The shock is usually fatal at an intensity of 400 volt of an interrupted or 1000 volts of a constant current. Jellinek states that a constant current of, on an average, 500 volts, and an interrupted current of 300 volts has a fatal effect, but he regards any intensity over 200 volts as dangerous under certain circumstances. His investigations show that a severe electric shock, like a stroke of lightning, produces as a rule organic changes in the nervous organs which can be demonstrated by microscopical examination.

Kurella (" Zwanglose Abhandl. aus d. Gebiet der Elektropathol.," etc., Leipzig, 1905) and M. Bernhardt (Berlin, 1906) have collected all the important data with regard to *professional accidents of telephonists*. Although such cases are often merely the result of the sound (Eulenburg) or the fright, the electric shock may sometime be the actual cause.

Neuroses may also develop after *surgical operations*. I have observed them specially after operations on the ear, but also after *perityphlitis* and *ovariotomy*, and it is difficult under such circumstances to determine how far the symptoms are due to the pre-existing disease, the absence of an organ, etc. Urbantschitsch (*Z. f. N.*, xxvi.) gives data as to the reflex paralyses which follow aural operations.

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[1] *Ärztl. Sachverst.*, 1901, and *B. k. W.*, 1905.

[2] *N. C.*, 1901. See also Mills-Weisenberg, *Univ. of Penns.*, 1903.

[3] " Elektropathologie. Die Erkr. durch Blitzschlag und elektr. Starkstrom," etc., Stuttgart, 1905; also *W. m. Pr.*, 1905, and " Path. Ther. und Prophyl. der el. Unfälle," *D. m. W.*, 1907.

[4] " La mort et les accid. par les cour. industriels," Genève, 1901-1902. See also Kleber, " Wie bekämpfen wir die uns durch die Elektrizität drohenden Gefahren ? " Berlin, 1905.

The symptoms sometimes appear immediately after the accident, but days, weeks, and even months may pass before they develop.

Pain in the affected part is usually the first complaint and is the chief subjective trouble during the whole course of the disease. In the neuroses which follow a railway accident, the pain is usually situated in the back, the sacral, or occipital regions. It gives rise to *constraint in the active movements*, as the patient endeavours to fix the painful parts and to avoid or, as far as possible, restrict the movements which would disturb them.

The pain is accompanied by other symptoms which are specially marked if the mechanical concussion has directly affected the brain (injury to the head, railway accidents), or if the accident has been associated with intense mental excitement. These phenomena are specially of a *mental* nature; *hypochondriacal-melancholic* depression develops, and is often manifested by the facial expression and outward bearing (Fig. 411). The patient gives way to gloomy thoughts about his misfortune, his illness, and his "hopeless" position. He is at the same time abnormally excitable and sensitive, weeps on the slightest occasion, and is effeminate in his demeanour. He very often complains of anxiety, restlessness, and dread, and has sometimes attacks of intense fear, less often of hallucinatory delirium. In most cases there is no real impairment of the intelligence, but the *memory* often becomes *weak*. The constant introspection and preoccupation with his own condition lessens the patient's interest in the outer world, and this may give rise to apathy which simulates mental weakness. Progressive dementia is very rare and occurs only in peculiar cases. Marked modifications in character may be a symptom of the psychoses [1] which develop from injuries to the head.

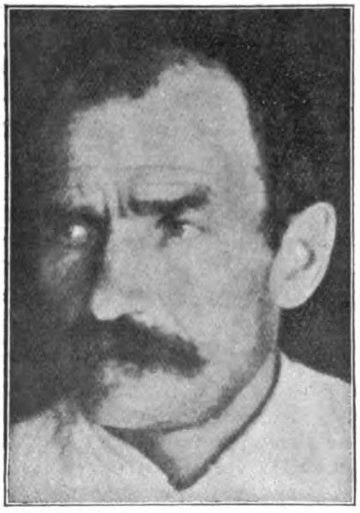

FIG. 411.—Facial expression in traumatic neurosis (hypochondriacal depression). Oppenheim.

Headache and *attacks of vertigo* are very prominent symptoms of the neuroses which result from head injuries. The giddiness may cause the patient to become pale and fall down. In many cases it comes on when-

[1] This is not the place to give an exact description of the traumatic psychoses (consult the work of Krafft-Ebing, Guder, Hartmann, Thomsen, Kaplan, Schäfer, Rathmann, Werner, Viedenz, Kalberlah, etc.). Krafft-Ebing, with whom the majority of other writers agree, distinguishes between *primary* traumatic insanity, which is the direct consequence of the trauma or the cerebral concussion, and the *secondary* form, which develops after a clear interval. There is no such thing as a well-characterised traumatic insanity, but some features are very noticeable. In the primary form dementia is the most marked symptom, but hallucinatory confusion and catatonic conditions also play an important part. The secondary form is definitely characterised by the degeneration of character, the irritability—Kaplan's explosive diathesis—and the tendency to vasomotor disturbances (congestion, intolerance of alcohol). Kalberlah emphasises the resemblance to the alcoholic psychoses (*A. f. P.*, Bd. xxxviii., with references to the earlier literature). Among the other contributions to the question see those of Reichardt, *Z. f. P.*, Bd. lxi.; Weber, *D. m. W.*, 1905; Heilbronner, *M. m. W.*, 1905; Phleps, "Psychosen nach Erdbeben," *Jahrb. f. P.*, xxiii.; Viedenz, *A. f. P.*, Bd. xxxvi.; Kölpin, Volkmann's "Samml. kl. Vortr.," Nr. 418, 1906. Transitory delirium in the course of the traumatic neuroses has been noticed by Oppenheim, Heveroch (*Casop. lek.*, 1904), and Pelz (*M. f. P.*, xxi.).

ever he bends down, and is then as a rule associated with marked flushing of the face and mucous membranes (conjunctiva, etc.), and sometimes with loss of consciousness. There may also be attacks of *complete unconsciousness*. It is well known that true *epilepsy* may be the result of injury to the head, and convulsions and mental attacks of an epileptic nature have been observed after railway accidents which did not involve any injury to the head (Westphal [1]). A combination of hysterical and epileptic convulsions after a head injury has been described, *e.g.* by Nonne.[2]

Injuries may also cause a definite and characteristic kind of epileptic attack in a reflex way; this is the so-called *reflex epilepsy* (see chapter on epilepsy). This should not be identified with *hysterical convulsive conditions*, which may also be a consequence of the injuries described. I can from personal observation assert that injuries to peripheral parts of the body, especially the foot, may result in attacks in no way differing from cortical epilepsy. The various forms of convulsion which are not accompanied by loss of consciousness, in particular *localised muscular spasms*, are prominent symptoms of the traumatic neuroses. *Convulsive tic* is specially common. Symptoms corresponding to myoclonia (*q.v.*) have occasionally been observed.

The symptoms mentioned so far show the *elements of mental disorders and of neuroses*, but in addition there are a number of others which we have learned to recognise as characteristic of neurasthenia and hysteria. Amongst the neurasthenic symptoms we include *insomnia*, general *muscular weakness* and *exaggeration of the tendon reflexes*, which are present in most cases. As a rule this exaggeration is general, but it may be limited to or most marked upon the side on which the other symptoms appear. Further, they include *increase of the mechanical excitability of the muscles and nerves*, and a number of *cardiac* and *vascular* symptoms. Acceleration of the pulse-rate is common, and abnormal excitability of the nervous system of the heart still more so, slight exertion and mental impressions giving rise to an excessively rapid pulse.

Consult also Jossilewsky, "Schwankungen der Pulsfrequenz bei verschied. Körperlagen," etc., Dissert., Berlin, 1905. "Orthostatic tachycardia" (Thomayer)—excessive acceleration of the pulse rate during the act of rising from the recumbent to the upright position—is merely a kind of manifestation of this instability, and naturally occurs also in the non-traumatic neuroses (Strauss, *Charité-Annalen*, 1904; Vaný sek, *Arch. bohém.*, vi.).

The pulse may be very small; it is sometimes notably large and soft, resembling the febrile pulse, and according to F. Müller the *sphygmographic curve* shows absence of the rise due to tidal waves, very marked and deeply situated dicrotic waves, with abrupt rises and sharp apices (dicrotic pulse). See also the remarks on p. 1121. Debove (*Presse méd.*, 1904) describes the occurrence of bradycardia, but his observation is not convincing.

Strauss (*N. C.*, 1901), on the evidence of investigations with Gärtner's tonometer made by Federn (*M. m. W.*, 1896), Schüle, Hochhaus, and himself, states that the blood pressure is increased, but this is contradicted by Hascovec (*R. n.*, 1903) and others. See also Bing, *B. k. W.*, 1906.

Irregularity of the heart's action is less common. Physical examination of the heart as a rule reveals no abnormality beyond the increased action, but the nervous affection may develop into an *organic* disease of the heart (dilatation and hypertrophy of the ventricles, arterio-sclerosis), as I have several times found in the traumatic neuroses, and as Strauss,

[1] *B. k. W.*, 1879; *Charité-Annalen*, 1880.

[2] *Mitt. aus d. Hamb. Staatskrank.*, viii.

Goldscheider,[1] and Leers,[2] have also stated. Some of the symptoms of exophthalmic goitre may also develop after injuries; but the whole syndrome of this affection is very rarely produced in this way. In the later stages *arterio-sclerosis* is frequently present. In traumatic neuroses of long standing accompanied by marked unilateral vasomotor disorders, I have seen the arterio-sclerosis develop earlier on the affected side.

Vasomotor disturbances are present in the majority of cases. These include cyanosis in more or less extensive areas of the skin, slight redness of the face, throat, thorax, and nape of the neck, and in particular the symptoms of *urticaria factitia* (*dermographia*, see Fig. 412).

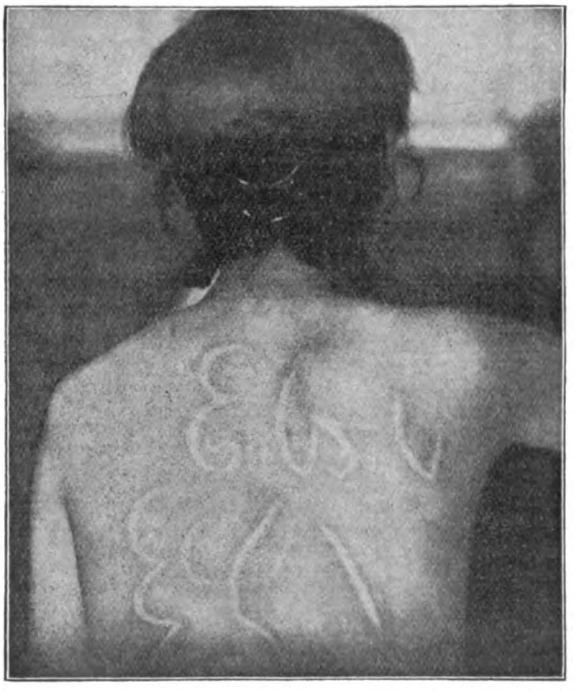

FIG. 412.—Dermographia.

The observations of Stursberg (*A. f. kl. M.*, Bd. lxxxiii.) certainly show that no great importance can be attached to this symptom, but from personal experience I am convinced that it is a pathological condition, although it may be the only manifest expression of the neuropathic diathesis.

I have found in one case in which the other vasomotor phenomena were limited to the right side of the body, that plunging the right hand into cold water brought on urticaria in that limb and caused dilatation of the pupil of the same side. I have certainly several times succeeded in demonstrating the vasomotor disorders which were not evident at the time of examination by holding the limb under cold water running from a tap.

Müller,[3] in a few cases with prominent labyrinthine symptoms, found hyperæmia of the tympanic membrane, and inferred from this that there was congestion in the inner ear; other writers, such as Baginsky,[4] have been unable to confirm this statement.

[1] *B. k. W.*, 1906.
[2] *N. C.*, 1906. See also Watermann-Baum, *N. C.*, 1906.
[3] *D. m. W.*, 1899.
[4] *B. k. W.*, 1905.

The forms of œdema described on p. 1090 are common signs of traumatic hysteria or neurasthenia. It is as yet uncertain whether the so-called hard, traumatic œdema (Secrétan,[1] Borchard,[2] Grünbaum [3]) may have a nervous origin. I have seen a few cases in which an œdema of this kind was the only persistent symptom of an injury to the limbs. Symptoms of *Raynaud's disease* have also been observed by myself and others.

Among the *trophic* disorders, we may mention premature greyness and loss of the hair (Stepp, Ladame). I have seen an alopecia which began on the anæsthetic side spread gradually over the hair of the whole body.

Heinicke (*N. C.*, 1903) has published interesting cases of change in the colour of the hair due to nervous affections. Although the observations which he has collected relate to the psychoses, they deserve consideration in this connection also. Reid describes an alopecia unguium.

The motor weakness is often accompanied by *tremor*, which corresponds either to a simple nervous type or to the various forms of hysterical tremor, or to the tremor of exophthalmic goitre, paralysis agitans, etc. It is usually increased by introspection and self-observation, and often completely disappears if the attention is distracted. In a few cases specially studied by Nonne [4] and Fürstner [4] (but which I had already considered), the chief symptom was shaking tremor which increased during movements, was accompanied with contracture of the muscles, and greatly impaired the power of standing and walking. *Fibrillary* tremor is present in many cases; it is either general in character or limited to the muscles of the paretic limb. In some cases it is only brought on by over-exertion. It may also be provoked by electrical stimulation (Rumpf [5]).

In addition to the inhibition of movement caused by the general muscular weakness and lack of motor energy, *paralysis* may be present. This differs so essentially from that caused by organic disease of the central nervous system, and resembles so completely the paralysis of *hysteria*, that it is regarded by Charcot and others as entirely identical with it.

In its *localisation*, the paralysis may, *e.g.* after a railway accident or contusion of the spinal region, correspond to the type of *paraparesis* or *paraplegia*. *Hemiparesis* or hemiplegia, almost always sparing the facial and hypoglossal, are more common. The paralysis is not infrequently limited to one extremity. The fact that unilateral paralysis *always develops on the side which has been affected by the injury*, is of great diagnostic importance. The functional hemiplegia which is caused by injury to the head therefore appears on the side of the trauma.

The paralysis is sometimes flaccid, but is more often associated with contracture, which resembles that accompanying hysterical paralysis and described under that heading. *A pseudo-contracture* is sometimes seen, *i.e.* a position corresponding to contracture, but without any muscular tension. Habit paralysis and contracture (see p. 413) have been observed chiefly in traumatic affections.

I have seen a few cases of contracture which in its form resembled none of the well-known types, and was associated with symptoms of motor-irritation, which suggested sometimes that of paralysis agitans, sometimes that of athetosis. I have also seen under these conditions the crampi musculorum, described by Wernicke (*B. k. W.*, 1904) as "Krampusneurosis."

[1] *Rev. méd. de la Suisse Rom.*, 1901. [2] *M. f. U.*, 1903. [3] *D. m. W.*, 1903.
[4] *N. C.*, 1896. [5] *D. m. W.*, 1890.

If the paralysis is incomplete, the effect of the obvious efforts made by the patient to move the limb may be slight. The reason for this is that the motor impulses are not properly distributed, but find their way into muscles which have nothing to do with the desired movement, or which may even by their contraction exercise an inhibiting influence upon it. This may give one the impression of being simulated (see below), but it has a pathological basis and is apparently due to a loss of memory of the distribution of the motor impulses necessary for the execution of a purposeful movement.

The paralysis is often *absolute*; on a casual examination one would say that the affected limb hardly seemed to exist for the patient. Sometimes, however, it is found that only *conscious, voluntary* movement is impossible, the muscles becoming active during emotion or from associative or reflex causes. Thus one of my patients, when on the point of falling down, held on by his hand, over which he had lost all voluntary control. Another patient who was unable to move his head when asked to do so, very often in speaking accompanied his words with unconscious movements of the head, such as he had formerly been accustomed to make. The limbs, though apparently paralysed, can often be forcibly moved during the excited stage of chloroform narcosis.

The limb directly affected by the injury is always most intensely paralysed.

The paralysis is not infrequently accompanied by *atrophy*, which as a rule is slight and associated merely with quantitative disturbance of the electrical excitability (simple diminution).

It should not be forgotten that slight differences in the size of the limbs of the two sides occur even in normal conditions; the right arm is apt to be somewhat greater in circumference than the left. Thus Rawitsch, according to a note of Bum's, found in 500 healthy soldiers a difference in size amounting to 4 cm. at the shoulder and to 1-1·5 at the fore and upper arm.

It is uncertain whether the acute bone atrophy described by Sudeck (see p. 65) should also be regarded as a symptom of nervous origin.

Disturbances of sensibility and of the special senses are very common, although not constant. Pain and paræsthesiae of various kinds are almost always present, and objective sensory disturbances are very common. It is an important rule that these are present either exclusively or mainly on the side of the body corresponding to the injury. Whilst hyperæsthesia is usually limited to certain sites, *e.g.* the skin over the injured part, the anæsthesia has usually a greater extension, spreading over one side of the body, the arm, shoulder and thorax, the arm and face, or more or less over the whole body. There is very frequently merely a *hyperæsthesia*, complete anæsthesia being much more rare. The sensibility to pain is specially apt to be diminished. It should be noticed that this analgesia or hypalgesia for the prick of a pin or the faradic brush may be associated with tactile hyperæsthesia of the same region.

The observations made by Voelker[1] after the Heidelberg railway accident are of great interest. He reports that the injured persons underwent most painful operations without any anæsthetic, and that setting a fracture, removing splinters, cleansing the wounds, cutting away their margins, suture of the bones, etc., were accompanied by no sense of pain.

[1] Bälz (*Z. f. P.*, Bd. lviii.), Finucane, and others have published interesting papers upon the symptoms directly following injuries accompanied by mental shock.

One patient stated that she felt the manipulations, but was conscious of no pain. Symptoms of paralysis and inco-ordination appeared in a few cases within a few hours or days after the accident.

The anæsthesia is, as a rule, of a mixed character or may involve the special senses. *Concentric narrowing of the field of vision* is an important sensory disturbance. Perimetric investigation is necessary to demonstrate this condition. It usually appears in both eyes, and where there is hemi-anæsthesia it is most marked on the anæsthetic side. It may be the only sensory disturbance, but is by no means a constant symptom. I used to find it much more frequently among the severe cases in the Charité than among my present patients at the Polyclinic.

Nervous buzzing in the ears and nervous deafness are not uncommon results of injuries to the head.

The syndrome caused by lesion of the labyrinth or the vestibular nerve, in particular the form of vertigo and disturbances of equilibrium corresponding or allied to Ménière's disease, is even more common. As a rule it is associated with diminution of the bone-conduction in the head and loss of the high notes, but it is very difficult to distinguish functional symptoms from those caused by an organic lesion (observations of Wanner, Gudden, Stenger,[1] Ziemssen, Gradenigo, Baginsky,[2] Krebs,[3] Rhese,[4] and the comprehensive papers of P. Bernhardt [5] and Passow [6]).

An injury to the left ear of a telephone girl caused by electricity was followed by clinical symptoms of a traumatic neurosis, vertigo and disturbance of equilibrium being very marked. Consult also M. Bernhardt and Kurella (*loc. cit.*). The latter has sought to explain the affections of the heart and respiration by assuming that the concussion is directly conveyed to the bulbar centres. Baginsky thinks that concussion of the labyrinth is usually associated with organic changes.

The anæsthesia or hypæsthesia is often accompanied by loss or diminution of the *cutaneous reflexes*, which is most apparent when the symptoms are unilateral. The relation between the condition of the reflexes and sensation is not, however, a constant one.

The various forms of *articular rheumatism* are very often due to a trauma.

In many cases the patient's power of locomotion is impaired, and various *disturbances of the gait* may arise, which are very difficult to explain. The gait is often affected by pain and stiffness in the back, and the pain may cause the patient to assume very peculiar positions and attitudes. The gait may, on account of the rigid position in which the legs are held, greatly resemble a spastic one, but the toes do not cleave to the ground, nor is there any special muscular rigidity in the recumbent position. I have already, in my first papers upon the traumatic neuroses, described this *pseudo-spastic* condition, which has been subsequently referred to by Fürstner, Nonne, Krafft-Ebing, Sommer, and others. The idea of being unable to walk may so influence the innervation of the muscles that a kind of stuttering in the legs may appear on walking (see section on abasia). A pseudo-ataxic disturbance of gait is less common; the legs are dragged, and the heels set down with a stamp, although no

[1] *D. m. W.*, 1905.
[2] "Die Unfallbegutachtung in der Ohrenheilkunde," *B. k. W.*, 1905.
[3] *Charité-Annalen*, xxvii.
[4] *D. m. W.*, 1906.
[5] *Vierteljahrschr. f. gerichtl. Med.*, xxv.
[6] "Die Verletzungen des Gehörorgans," Wiesbaden, 1905. See also Bárany, *B. k. W.*, 1907; and Bárany, "Vestibularerkrankung und Neurose," *N. C.*, 1906. We may also refer to the article of Urbantschitsch already mentioned, and to that of Frey-Fuchs (*Obersteiner*, xiii.) upon reflex epilepsy of the ear.

ataxia can be detected when the patient assumes the recumbent position, and the disturbance is quite different from cerebellar ataxia. Walking is associated with very marked tremor in the legs, which increases in intensity with every step.

Speech is not infrequently involved. Mutism may develop directly after the accident. Symptoms allied to *stuttering* and stammering are frequent, but syllable-stumbling is seldom observed. *Paralysis of the vocal cords* (adductor paresis) has so far been noted only in a few cases.

The reaction of the pupils to light is usually retained, and indeed often exaggerated (Oppenheim, Hübner). *Loss of this reflex* is in our experience quite unusual. Even although the cases in which it does occur correspond in all their other symptoms to the type of the neuroses, there can be no doubt that a *complication* with organic changes of the nervous system is present. *Inequality of the pupils* is often observed, but this sign is only of pathological importance when it is very marked and there are no differences of refraction between the two eyes. The inequality may only be discovered in a dark room or when the eyes are shaded. The dilated pupil is usually on the side in which the pain, sensory disturbances, and symptoms of paralysis are present. Nystagmus seldom occurs, but I have in a few cases observed a tremor of the eyeballs during attempts at movement, associated usually with blepharoclonus (see p. 1088). Apelt [1] has lately published similar observations. In two cases in which the patients complained of headache and giddiness, I have seen transient exophthalmus produced by stooping.

True paralysis of the eye muscles is not one of the symptoms of traumatic neuroses, but a motor disturbance like that in hysteria, generally due to contracture, may develop here also, as I have seen, and as A. Westphal (*D. m. W.*, 1905) and others have described.

In the rare cases of traumatic neurosis which show *atrophy of the optic nerves*, a complication has been present, the injury having given rise simultaneously to functional and organic changes.

Gastric disturbances, such as anorexia, vomiting, and profuse diarrhœa, occur only in a small proportion of cases. The *state of nutrition* may be severely impaired, the loss of physical strength being very striking, but for the most part it remains normal: indeed, it is not unusual for the patient to increase in weight during the illness.

The patient often complains of *difficulty in micturition*, constipation, and impotence—symptoms which usually cannot be confirmed by objective examination. *Polyuria*, *albuminuria*, and *glycosuria*, are uncommon symptoms, but traumatic diabetes may be associated with or develop after the traumatic neurosis (Ebstein). Senator has carefully studied the relations between trauma and diabetes. Intercurrent *attacks of fever* have appeared in a few cases. According to the investigations of Mann, resistance to galvanism is lessened in the skin of the head in the cases with headache, vertigo, noises in the ears, and other symptoms of a similar kind.

The division of the traumatic neuroses into a *general* and a *local* form, which Strümpell suggests, has some justification, but there is no essential difference between them. The former is characterised by signs of a general affection of the nervous system, whilst in the latter symptoms localised to the injured part of the body are most prominent.

[1] "Ärztl. Sachverst.," 1903.

Finally, it should be remembered that an injury may simultaneously give rise to an organic disease and to a neurosis, and therefore to a blending of the symptoms of these two different forms of disease.

Friedmann [1] saw injury to the head followed by symptoms which he associated under the name of " vasomotor syndrome." These include headache, giddiness, congestion, vomiting, intolerance of alcohol, etc., associated possibly with symptoms of paralysis of the cranial nerves (?), and in rare cases with febrile attacks. In a few of these cases the finer cerebral vessels were affected. These conditions undoubtedly often follow injuries to the head, and are sometimes associated with the symptoms of the functional neuroses.

The above description applies to the *severe* forms of traumatic neuroses. But there is a large and apparently steadily increasing number of cases in which the majority of the symptoms just described are absent, and in which the subjective symptoms, the headache, loss of strength, etc., of which the patient complains, are accompanied by few if any objective signs. The cases in which no objective symptoms can be found are often not those of a nervous disease, but of a hidden surgical affection. Freund has demonstrated this by a number of examples, and we have specially learned to recognise these cases since the introduction of X-ray examinations. The great majority of cases of simulation also belong to this class. Some writers (Einstein, Jessen, Saenger) are of opinion that cases of traumatic neuroses have become gradually less common during recent years.

Pathological Anatomy.—The term neurosis in itself implies that hitherto no pathological basis has been discovered for this condition. We assume that the functional disturbances are produced by molecular changes in the central nervous system. It is true that a very small number of cases have been examined post mortem, but in these the result was usually a negative one. A few observations prove, however, that concussions which have not resulted in a direct, severe lesion of the central nervous system, may lead to disease of the *vascular system of the brain*, especially the smaller vessels, to arterio-sclerosis, hyaline degeneration, and endarteritis obliterans, and it is probable that some of the symptoms mentioned, such as the persistent headache, the attacks of giddiness, and the vasomotor symptoms may in some cases be due to such changes (Kronthal,[2] Friedmann,[3] Köppen), although it is not impossible that the former may be the result of repeated vasomotor disturbances. Schmaus has shown that concussion of the spinal cord, although not associated with gross pathological lesion of the organ, may cause destruction of the nerve fibres or swelling and degeneration of the axis-cylinders, disintegration of the myelin, etc. Bikeles has also shown that a blow upon the head of an animal may produce disintegration of the myelin in the nerve fibres of the medulla oblongata and the spinal cord, which can only be shown by Marchi's method. Kirchgässer [4] and Kazowsky have also found this, whilst the papers of Scagliosi [5] and Luzenberger relate specially to the conditions in the nerve-cells (which moreover are of doubtful value). These results of investigation show the necessity for caution in the diagnosis, and are specially adapted to throw light upon the symptoms

[1] *A. f. P.*, xxiii.; see also his latest paper, *N. C.*, 1906.
[2] *N. C.*, 1889 and 1890.
[3] *A. f. P.*, xxiii., and *Z. f. N.*, xi.
[4] *Z. f. N.*, xi.
[5] *V. A.*, Bd. clii.

which are not of a psychogenic nature. Some recent writers—Vibert, Knapp, Crocq, and others—express their belief that fine organic changes occur in the traumatic neuroses. It is obvious that severe functional disturbances, such as may be produced by a stroke of lightning or an electric shock, are due to organic processes (see above).

Pathogenesis.—According to our conception the traumatic neuroses are the effect of *mental* and *physical* shock. This affects chiefly the cerebrum, and produces molecular changes in the areas which control the higher mental functions and the motor and sensory functions related to them. This view does not exclude the possibility that fine organic changes (in the vessel walls, disintegration of the myelin in single fibres, etc.) may exist and form the basis of individual symptoms. A trauma affecting some peripheral part of the body may also act upon the cerebrum; the shock may be conducted directly to it by the sensory nerves and produce similar changes in it, or a persistent irritation may be conveyed to it from a cicatrix.[1] This interpretation which I have given has been accepted by Goldscheider and by Binswanger; the former has published some very interesting observations and investigations which show that these functional disturbances continue during narcosis, or may even become then evident for the first time, a fact which cannot be brought into accord with the assumption of their auto-suggestive origin and mode of production. Local injuries are specially liable to have this effect if the cerebrum is already abnormally sensitive (neuropathic disposition), or if it has been already affected by the accident itself (fright, excitement). Goldscheider agrees with me in this conception.

This theory is opposed by Charcot, who thinks that traumatic hysteria is produced by means of *auto-suggestion.*

Strümpell has lately stated that the idea of making money out of his accident, the desire to acquire an income, and the knowledge that he can claim it, etc., are important, if not the chief, causes of the neurosis. Although this factor may be concerned in so far that the non-satisfaction of his claim and his consciousness of a grievance give rise to depression and irritability, and that his anxiety about obtaining compensation increases his introspection, I cannot admit that the symptoms are produced in this way. This is contradicted above all by the fact that traumatic neuroses may occur in individuals who have no claim for compensation, as has been shown by a great number of cases observed by myself, and in particular by P. C. Knapp (*Br.*, 1897) and Döllken (*N. C.*, 1906). In a few very severe cases under my care the trouble had developed, *e.g.* in school-boys or young girls, after a fall on the occiput in skating; in one girl of ten it followed a railway accident.

Many recent writers, such as Nonne (*M. f. Unf.*, xiii.; *Ärztl. Sachverst.*, 1905), Quincke, Hoche (abstract, *C. f. N.*, 1907), Windscheid, Hellpach (*N. C.*, 1906), have expressed more or less definitely their agreement with Strümpell's opinion. Gaupp (*M. m. W.*, 1906) is inclined to ascribe much greater importance to the desire for compensation and to the social factors than to the injury itself.

Compare on the other hand the interesting statistics of Merzbacher (*C. f. N.*, 1906) and Döllken. I must confess that, although fully recognising these accidental factors—which have led to the term *compensation-hysteria*—I believe in *the existence of true traumatic neuroses quite independent of these factors.*

Diagnosis.—The chief difficulty consists, not in the differentiation of the traumatic neuroses from other diseases of the nervous system, but in determining whether we have to do with disease or simulation. As a workman who is injured in the course of his work knows that he has a claim for compensation, it is not unusual for him to represent anything that may be the matter with him as the result of an accident. The frequency of simulation in traumatic neurosis has previously been greatly

[1] I might mention in passing that slight injuries may exert a curative influence upon certain neurasthenic troubles. I have known two cases in which persistent and evidently neurasthenic constipation, which had lasted from childhood, disappeared after a fall which caused some contusion. In another case hemicrania, and in a third nervous asthma, which had persisted for about 15 years, were cured after an accident. It is well known that injuries, especially those causing suppuration, may have a favourable influence upon the psychoses; this has again recently been emphasised by Bach.

over-rated, as the nature of this condition has been little understood and the patients have been examined by those who had no psychiatric training. The fact that the same clinical conditions have been observed after injury in different countries and races is in itself a proof that we have to do with a real disease. Still more is this shown by the fact that precisely the same clinical conditions may follow injury in cases where there is no question of compensation. However, the possibility of simulation, and especially of exaggeration, should always be borne in mind in making the diagnosis. It is highly advisable not to enter upon an examination with a pre-formed opinion, but to make a thorough examination as in every other case. It must be our endeavour to discover any objective symptoms of the disease. Chief among these are—

Persistent exaggeration of the tendon reflexes, of the mechanical excitability of the muscles and nerves, fibrillary tremor or *tremor* and *clonic twitchings* in certain muscles in which a healthy person cannot produce independent tremor (*e.g.* triceps, sup. longus, muscles of the shoulder-blade, omohyoid, etc.), *atrophy, vasomotor phenomena, symptoms of cardiac neurasthenia, disorders of secretion, inequality of pupils*, etc. The latter, it is true, is occasionally present in healthy persons, but so seldom that it is hardly of importance. My experience is that *typical narrowing of the field of vision* cannot be simulated. Other writers (Charcot, Wilbrand, König,[1] Arnheim) have expressed a similar opinion and have shown that slight degrees of contraction cannot be simulated without practice and expert knowledge. They (as well as Peters [2]) consider 5 to 10° of contraction of the field of vision from the periphery unaccompanied by any errors of refraction, hypermetropia in particular, to be a pathological condition. Schmidt-Rimpler takes the opposite view and thinks it is necessary to make a *campimetric examination* at different distances, *i.e.* to project the field of vision upon a surface, *e.g.* a board, and thus to make measurements at various distances. The visual angle must naturally appear to increase in extent the farther away the measurement is made, whilst the person who is simulating and is ignorant of this fact does not allow his field of vision to increase in proportion to the distance. Such a fact would be evidence of simulation if the patient were not mentally affected. It has, however, been shown by Oppenheim, Wollenberg, and others that this is not always the case in hysteria, and Greef [3] believes that a " tubiform contraction of the field of vision " may even be regarded as characteristic of hysteria.

On this question see Bach, *Z. f. Aug.*, xiv. Foerster's type of displacement (Verschiebungstypus) is a condition in which the test object, when moved into the field of vision from without, is seen farther towards the periphery than when it is moved in the opposite direction. The test object is first of all brought gradually from the periphery into the visual field, and thereafter from the centre towards the periphery; during the first test the point at which it becomes visible is marked, and during the second the point at which it vanishes. Two fields of vision are thus obtained, the former of which is larger in every direction than the latter. According to the investigations of Wilbrand,[4] Koenig, Placzek, and others, this symptom often occurs in persons suffering from traumatic neurosis, and, as Peters admits, it can hardly be simulated; others (Schmidt-Rimpler,[5] Peters, Voges), however, maintain that the symptom may also be found in healthy individuals, so that the test is not a decisive one. Wilbrand states that the enlargement which the visual field shows in a dark room, takes place more slowly under pathological

[1] " Uber Gesichtsfeldermüdung," etc., Leipzig, 1893, and *Z. f. N.*, vii.
[2] *Z. f. N.*, v.
[3] *B. k. W.*, 1902.
[4] *Jahrb. d. Hamb. Staatskrank.*, 1889.
[5] *W. m. W.*, 1895, and *D. m. W.*, 1892.

conditions, but this fact and the method founded upon it, of measuring the visual field in a dark room (see p. 75), are too complicated to be of any practical use.

Reuss (*W. kl. R.*, 1902) has described another method for perimetrical examination of the condition of exhaustion. See also Kroner, *Z. f. kl. M.*, Bd. liv.

Investigations by Wolffberg (*A. f. Aug.*, 1903), Frankl-Hochwart, and Topolanski [1] point to certain relations between contraction of the visual field and quantitative diminution of the senses of light and colour. It is possible that further investigation may render these facts more valuable in determining the nervous disturbances of vision in traumatic neuroses, and in deciding whether the condition is real or simulated.

We cannot here discuss the various methods for discovering simulation of unilateral or bilateral blindness, deafness, etc. Most of them are of little importance and value as compared with the blindness and deafness of *hysterical* or *psychogenic* origin; and so is the fact ascertained by using the prism and stereoscope, that the patient can see with the eye which he pretends is blind, and the discovery by similar tests that his deafness is simulated. If, for instance, a tuning-fork is placed on the patient's head and it is found that the note is heard louder when the ear said to be deaf is closed, this proves that the patient can really hear with it. Each ear should be connected with a tube which opens into a mouth-piece into which one can whisper; the tube leading to the sound ear may then be suddenly closed and opened again by compressing it without the patient's knowledge. He should then be asked to repeat what is whispered into the tube, and in this way one can discover whether the one-sided deafness is actual or simulated. Another test is to have two persons whisper simultaneously into the two ears words of the same number of syllables and in the same measure, which the patient is asked to repeat. Binswanger and Krause (*M. f. P.*, vi.) have reported a number of such tests. It should not be forgotten, however, that there is a form of blindness and deafness in which the patient sees and hears without being conscious that he is doing so. It may therefore be possible by such tests to convict him of simulation or perhaps to cure him, *i.e.* to free him from his symptoms; but we should beware of regarding this fact as in itself a proof of simulation. Certainly circumstances may occur whilst we are thus testing the patient, to show that he cannot be trusted and is consciously pretending. Facts and advice worthy of attention will be found in the writings of Freund and Sachs. See also Hasslauer, *D. milit. Zeitschr.*, 1903; Hechinger (*Z. f. Ohr.*, Bd. li.) upon Gowseef's brush-test; Leupoldt, "Nachweis der Simulation von Taubstummheit durch Schreckwirkung auf akust. Reize" (Sommer's "Klinik," etc., i.); Wernicke, "Obergutachten," etc. (*M. f. P.*, xvii., Ergänz.); Groenouw and Stenger (*D. m. W.*, 1907); Sand, "La Simulation," etc., Bruxelles, 1907.

A thorough knowledge of the psychoses is necessary before one can make a diagnosis of mental disorders. Of late years psycho-physical examinations have been called into use, more especially the methodical measurements of mental work employed by Kraepelin, Gross, Sommer, Roeder, Specht,[2] Leupoldt,[3] and Plaut,[4] to determine the conditions of fatigue, distraction, etc. By these means we may obtain evidence which excludes simulation.

Though constancy of the symptoms is very convincing evidence of their reality, it should not be forgotten that the symptoms of the neuroses are subject to great variability, so that the results of tests made at different times are not necessarily identical.

Finally, it should be specially kept in mind that although any or all of the symptoms may be simulated, it is hardly possible to simulate the whole clinical picture.

Jaksch [5] points out that so-called *alimentary glycosuria* may be an objective symptom of the traumatic neuroses, as the use of about 100 gr. of grape-sugar produces transient glycosuria, which becomes evident

[1] *Beitr. z. Augenheilk.*, 1895. [2] *M. f. P.*, xv.; *A. f. die ges. Psych.*, iii.
[3] "Die Untersuchung der Unfallnervenkranken mit psychophys. Methoden," *Klinik. f. psych. u. nerv. Krankheiten* (Sommer), i., Halle, 1906.
[4] *N. C.*, 1906. [5] *Verhandl. d. xiii. Kongr. f. inn. Med.*, 1895.

within two to six hours. But as this symptom occurs under other conditions and is by no means constant in neuroses of traumatic origin, it is doubtful whether it is of real diagnostic importance (Strauss, Arndt,[1] Strümpell,[2] Oordt, and others). Hoedke has found glycosuria the day after the accident in 60 per cent. of his cases.

In one of my cases hyperæsthesia in the injured thumb, of which the patient complained, was proved objectively by the fact that at this part, even with the weakest currents, the CC, AO, and AC gave the sensation of a blow or pain. The closing and opening of the current were of course managed so that the patient was unaware of them.

An important point in the diagnosis is the fact that the symptoms often extend over the *whole side of the body* involved in an injury. If, for instance, the arm has been injured and shows a condition of contracture and paresis, the *cyanosis* and *atrophy* which appear in the limb may cause a certain amount of suspicion, as both these symptoms, if not very marked, may be attributed to the inactivity, which may also be simulated. But, if we examine the leg which the patient moves normally and about which he has made no complaint, we not infrequently find the same symptoms there, though to a less degree. If the patient stands for a long time, the leg of the affected side alone becomes *cyanosed* and feels colder than the other.

In *testing the sensibility*, all suggestive influences should be carefully avoided.[3] Instead of asking the patient as to his sensation, one should first examine the *reflex* and *defence movements*, especially to painful stimulation. This may be of great value in unilateral sensory disorders in which the unaffected side can be used for comparison. If it is found on repeated examination that the reflexes are always weaker on one side than on the other, or if they are absent altogether, there is every probability that a sensory paralysis is present. The various forms of stimulation should then be used, and the patient's answers noted. By varying the strength of the stimulus and comparing the sensitiveness at the various areas where it is ordinarily slight with others which are more sensitive, it is possible to test the patient's good faith. If, for instance, there is slight tactile hypæsthesia, this will be specially evident as regards light touches with the brush, whilst firmer touches will be felt. Or, should there be hypæsthesia of the leg, the stimuli will not be felt at all on the soles or the balls of the toes, where in health they are but slightly felt, and at other places they will be less intensely perceived than on the other side. Examination of the temperature sense by Goldscheider's method (see p. 48) is also well adapted to test the patient's truthfulness, but it requires a great deal of time.

A method of examining the sensibility on the crossed and folded hands and fingers, which is described by E. Müller (*B. k. W.*, 1903) and founded on observations by Henri, may serve to differentiate psychogenic anæsthesiæ from those of organic origin, but not to detect simulation.

The results obtained by Biervliet and Ioteyko-Stefanowska (*Journ. de Neurol.*, 1903) are not of much practical value.

Gross contradictions in the statements of the patient render the results of examination useless. They may afford direct proof of his untruthful-

[1] *Z. f. N.*, x. [2] *B. kl. W.*, 1896.

[3] This precaution, which we have exercised for many years (see earlier editions of this textbook, etc.), has lately been indicated by Babinski (*R. n.*, 1908) as essential in determining the sensory disturbances of hysteria, etc.

ness, but we must remember that contradictions in the answers to such tests are very common, even in persons suffering from an organic disease of the nervous system (tabes, etc.), so that one should be very careful before assuming them to be a proof of simulation. We should also bear in mind that testing with the prick of a pin is not identical to testing with the electric current. The results obtained from examination with these methods need not of necessity agree with each other. Painful stimuli which affect the patient suddenly and unexpectedly at some insensitive (analgesic) spot, may provoke a violent movement of defence, as the suddenness of the shock—even when felt merely as a touch—causes fright.

Sommer and his pupils (Leupoldt, *loc. cit.*) have endeavoured to measure the reaction of fright to sound stimuli, etc., with suitable apparatus, and to use the plethysmographic method in this examination.

It cannot always be determined beyond doubt whether the patient is actually suffering pain or merely pretending to do so, especially as pain, like all the other symptoms of the neuroses, is influenced by attention and introspection and diminished when the attention is otherwise occupied. The data of physiologists (Schiff, Lombroso, Mantegazza, etc.) as to the effect of pain upon the action of the heart do not agree. It would seem that slight pain has an accelerating, and more intense pain a retarding effect upon the frequency of the cardiac contractions, but nothing can be definitely inferred from the investigations published. Egger, who used the faradic current, employing stimuli of weak and medium intensity, never found anything but acceleration of the heart-beat, even although there was no muscular activity during the examination. If points sensitive to pressure are present, pressure upon them produces not merely pain but marked increase in the pulse rate (Mannkopf's symptom), which may also be caused by the affected part of the body being placed in a painful position or attitude (Oppenheim). But it should always be remembered, as Egger's[1] careful investigations show, (1) that muscular movements which accompany the manifestations of pain may increase the action of the heart, and (2) that some persons, according to the observations of Tarchanoff, Van de Velde, etc., can accelerate the beat of the heart by a voluntary impulse (or by directing attention to the heart). In making these investigations care should be taken that the patient avoids muscular activity and forced respiration, and that his attention is distracted. Egger recommends the sphygmograph for these tests. In some cases I have been able to bring on vasomotor phenomena, *e.g.* flushing of the corresponding side of the face, by causing pain. Bechterew has independently noted this symptom. The pain may also be caused by painful movements and by the tremor which only accompanies these. These factors, to which we have repeatedly called attention, have since then been carefully studied by Erben. On the other hand, I[2] have sometimes found that very strong currents from the faradic brush applied to the

[1] *A. f. P.*, xxi. See also Martius, "Über die Lehre von der Beeinflussung des Pulses und der Atmung durch psych. Reize. Beitr. z. Psych. und Philos.," i., fourth edition, Leipzig, 1905; Jossilewsky, "Schwankungen der Pulsfrequenz bei verschied. Körperlagen," etc., "Inaug.-Diss.," Berlin, 1905; Colucci, *R. n.*, 1906; Rumpf., *M. m. W.*, 1907. It is still doubtful whether the "psychogalvanic reflex-phenomenon" can be used for investigating disturbances of sensibility. See Veraguth, *M. f. P.*, xxi.; Peterson-Jung, *Br.*, 1907.

[2] "Der Fall N.," etc., "Über einen Fall von traumat. Neurose." Vorlesung 1895-96, Berlin, 1896.

sensitive side of the body, greatly quickened the pulse beat, but when applied to the insensitive side they caused no appreciable change. H. Curschmann [1] found this also with regard to the blood pressure. But the negative results of these tests, the absence of Mannkopf's symptom, etc., should not of themselves alone be regarded as proof of simulation. I think the methods recommended by Fuchs and others for the detection of simulated tremor are impracticable. Erben regards the tremor as simulated if, under prolonged observation, the tremor movements become gradually coarser and slower, and signs of fatigue thus become manifest. The tremor is probably genuine if, when some fingers are artificially fixed, the others continue to tremble.

The sign described by Hösslin (see p. 1082) is not a certain indication of simulation, as it also occurs in hysteria. Zuckerkandl and Erben (*W. kl. R.*, 1903) point out that in forceful movements of a proximal part, *e.g.* the shoulder or elbow-joints, against resistance, the distal part (the wrist-joint) is instinctively fixed; in cases of simulation this does not take place. This symptom cannot be applied in the diagnosis of psychogenic paralyses.

In paralysis of the levator anguli scapulæ the power of gradually lowering the passively raised arm is impaired; if therefore this power is retained there can be no true paralysis of the abductors, but this does not exclude the possibility of psychical paralysis. Few of the other tests described by Erben (*W. med. Pr.*, 1906) for the detection of simulation of nervous disorders are of any value.

If dorsal flexion of the hand, though possible, does not take place when the patient is asked to grasp the physician's hand firmly, or if the force does not increase upon dorsal flexion, the case is probably one of simulation. This is not an absolute sign of simulation, nor is the sign of incomplete clenching of the fist described by Kaufmann, as it may on the one hand be a result of mental inhibition, and on the other a phenomenon due to the kind of manual work in which the patient has been employed (Thiem). The points regarded by surgeons as pointing to simulation of stiff joints do not apply to hysterical contractures.

Many distressing symptoms, *e.g.* the common one of nervous tinnitus aurium, are not accessible to objective examination.

The cases in which the subjective complaints of the patient do not correspond to any objective signs present much difficulty. The physician must not be afraid in such cases to pronounce his "*non liquet.*" Another very disturbing factor is that nervousness (and, according to Saenger, arteriosclerosis also) is becoming more and more common in working people, so that the objective signs of this kind may have been in existence before the accident, and may yet be mistaken, even by an observant physician, for symptoms of traumatic neurosis. It is difficult to avoid this error, but this matters less, since in typical cases the symptoms show some local relationship with the site of the injury.

It is dangerous to make the examination bear directly upon the detection of malingering. In the first place one should try to prove the existence of the disease. It is only when the result of examination is negative or absolutely contradictory and suspicious that one is justified in directly searching for evidence of simulation. In doubtful cases it is always advisable to have the patient under observation in a hospital where the superintendent is expert in the study of the neuroses and psychoses. The sleep and the paroxysmal symptoms (conditions of anxiety, spasms, attacks of giddiness) can there be properly watched. We agree with Freund, however, that it may be a most difficult and troublesome task to obtain convincing proof of the simulation of a neurosis.

[1] *Therap. d. Geg.*, 1906.

We might here refer to the very extensive literature on *simulation* of *mental diseases*, and simulation by those who are insane, in Fürstner (*A. f. P.*, xix.), Moeli ("Über irre Verbrecher," Berlin, 1888), Bolte (*Z. f. P.*, Bd. lx)., Jung (*Journ. f. P.*, ii.), Schott (*A. f. P.*, Bd. xli.), Bresler, ("Die Simulation von Geistesstörung und Epil.," Halle, 1904), and others.

Difficult as it is to determine whether we are dealing with disease or simulation, it may be just as difficult to decide upon the patient's *degree of earning capacity*. Exaggeration is frequent in these cases, but this may be a pathological condition (the patient really over-estimating his symptoms) or it may be artificially produced by the fact that the injured man has already been met with suspicion.

If the disorders are purely *local*, the earning capacity is usually merely limited. But even when there is only paralysis of one arm or leg, the general condition has to be considered, as a general neurasthenia may be present in addition to the local symptoms. If the condition corresponds more or less completely to neurasthenia, the power of working depends upon the severity of the condition. In slight cases work is usually a remedy, but a profound neurasthenic is generally quite unable to work, or he can do only the lightest tasks. This is much more the case as regards labourers than the educated classes, shopkeepers, etc. It applies also to the severe forms of hysteria.

A patient is not necessarily capable of working because he can use all his limbs. We have to consider how work affects his condition, whether he becomes quickly exhausted, and whether movements cause him pain. In order to determine these points it may be necessary to make the patient work and to note the condition of his pulse, respiration, etc.

We should do well in doubtful cases to over-estimate rather than under-estimate the limitation of the patient's working powers, and also to be very guarded in giving the verdict of complete inability to work. Above all, this inability should never be regarded as of a chronic nature.

It hardly needs to be said that we should ascertain whether the nervous condition may have been present before the injury to which it is wrongly attributed. *Alcoholism* in particular may produce symptoms identical in many points with those of the neuroses and psychoses. The presence of alcoholism is not, however, a proof that the existing condition is due exclusively to this intoxication. It is more probable that it increases the predisposition for the traumatic neuroses, so that some slight trauma (especially *injury to the head*) may give rise to a grave nervous disease. It is not the duty of the physician to declare that the patient has forfeited his claim for compensation on account of his indulgence in alcohol; he has merely to ascertain the facts, to state that although alcoholism is present the injury has been the exciting cause of the nervous disease, etc. Evidence of the traumatic origin of the affection can usually in such cases be furnished by the local symptoms produced by the injury (cyanosis, atrophy, local anæsthesia).

As regards the *differential diagnosis*, there may under certain conditions be some difficulty in distinguishing a traumatic neurosis from hæmatomyelia, spinal caries, brain abscess, and other affections. Such an error can usually be prevented by careful examination and observation.

Prognosis.—In slight cases there may be complete recovery, but even the neuroses in which the symptoms are chiefly local may be very persistent. The position of the patient, his struggle for compensation, his desire to make money, the instigations of relatives and unscrupulous

lawyers, and premature resumption of full work are all factors which influence the course unfavourably.

The more the *mind* is involved, the less favourable is the prognosis in general. If the cardiac and vascular symptoms are very marked, and dilatation of the heart and arteriosclerosis have developed, there is hardly any prospect of complete recovery. The prognosis is further affected by the fact that the neurosis frequently develops into a *psychosis*. I have seen this take place in a few cases under my own observation, *e.g.* one described by Goebel. The not inconsiderable number of suicides in traumatic neuroses reported in recent literature are mainly due to severe mental derangement. I have sometimes seen, even in cases which originally showed only local symptoms, a change for the worse occur to such a marked degree that ultimately one might term the condition a "derangement of the nervous system."

The prognosis is much more grave in nervous affections caused by electric shocks or discharge of strong currents, as the nervous system has usually been subjected to more or less severe organic lesions, quite apart from the fact that such shocks are frequently the direct cause of death (see the works of Jellinek and others already mentioned).

Treatment.—One must try from the first to prevent the development of hypochondriacal ideas, and to convince the injured man that neglect of his nervous troubles is the most effective means of getting rid of them. If one is certain that no serious disease is present and that the symptoms have merely grown out of the patient's morbid introspection, it is advisable to send him back immediately to work. In other cases he must be given time to recover, and he should not be compelled to resume *full* work before he is ready for it. This by no means implies that he should remain resting until his health is completely restored, as physical work (gymnastics) carefully carried out under medical supervision may be a most useful factor in his treatment. It is true that work of a productive nature, which brings in some reward, is of more real service (Bruns), and the advice given by Herzog, Strümpell, Auerbach,[1] and others, that a register should be organised for those capable of only partial work, deserves full attention. The principle has already been carried out in some places, *e.g.* Leipzig.

Forcible remedies should never be employed. The attempt, for instance, to reduce contracture by force always leads to the condition becoming worse. Applications of strong faradic brush currents for controlling the tremor, spasm, etc., should also be avoided.

In severe cases of general nervousness with depression, irritability, etc., a change of air, especially to the country, may have a good effect. Cold water treatment may also be of service. Bathing treatment in Kudowa, Nauheim, and Oeynhausen have been recommended. I have seen improvement in many cases and recovery in some after the use of the galvanic current. Galvanisation of the brain is specially recommended, and when there is stiffness of the back the current should be applied to the spinal region. This treatment may be continued for a considerable time, but I do not think it good to prolong it for more than a few months.[2] The faradic brush current sometimes cures the anæsthesia. Paralytic

[1] *Med. Klinik.*, 1906. See also the discussion in the *B. k. W.*, 37, 1907.

[2] See the inquiry into the treatment of severe traumatic neurosis in the *Ärztl. Sachverst.*, 1906.

conditions, if not associated with contracture, may indicate the use of the induction or labile galvanic current. Mild massage is also good in such cases and when there is stiffness of the muscles and joints. Excellent results are sometimes obtained in the medico-mechanical institutions.

If cortical epilepsy is present, it is necessary to excise the cicatrix which gives rise to the irritation, but this by no means always leads to recovery.

Treatment by drugs is of little use ; it is similar to that of the general neuroses. *Mental* treatment is by far the most important factor in these cases. The greater the discernment which the physician brings to bear upon these patients and their troubles, the more successful will he be in curing or improving their condition.

As regards *prophylaxis*, we would refer to the papers by Jellinek, Eulenburg, Kurella, Bernhardt, and Kleber. See also Mittelhäuser, "Unfall und Nervenerkrankung.," Halle, 1905 ; Stolper, *Vierteljahrschr. f. ger. Med.*, Bd. xxxi.

Among the traumatic neuroses we may perhaps include the exceedingly distressing sensations "in the absent limb" which sometimes follow *amputations* (Ambroise Paré, W. Mitchell, Pitres,[1] Charcot, Reny,[2] etc.). The patient has pain and various sensations (illusory sensations or movements, or psychomotor and psychosensory illusions according to Pitres), which he localises in the amputated extremity. These may develop directly after the operation or later. Any sensory irritation, or in particular any irritation or electrical stimulation of the stump of the amputation, may provoke these sensations. The limb is usually felt as if it were smaller and the stump nearer (Borek). The condition is exceedingly persistent. One of my patients, an Australian, had consulted the surgical authorities of many countries, and had in vain undergone many operations in order to get rid of a distressing sensation as if his amputated hand were spasmodically clenched.

As these symptoms are undoubtedly due to central processes, which have no organic foundation in the central nervous system, but are due to a peripheral lesion, we must include the condition among the traumatic reflex neuroses. This view has been adopted in some of the more recent works (Briele,[3] Gulbenkian [4]).

Hemicrania (Migraine)

Literature in the articles by Liveing, "On Megrim," etc., London, 1873; Thomas, "La migraine," Paris, 1887 ; Möbius, "Die Migräne," Nothnagel's "Handbuch," xii. ; Bioglio, "Contributo allo studio clinico dell' emicrania," Roma, 1905.

This very common affection occurs principally in persons of a neuropathic predisposition. It is often *directly inherited*—in 90 per cent. of the cases, according to Möbius. Observations have been published of cases in which the migraine was transmitted through four generations, or appeared in eight members of one family, etc. It usually first occurs at the age of puberty, and not infrequently in early childhood, or it may develop

[1] *Ann. méd. psychol.*, 1897. [2] *Thèse de Nancy*, 1899. [3] *Z. f. Chir.*, Bd. lxv.
[4] "Hallucinations du moignon." *Thèse de Paris*, 1902. See also the note by Souques-Poisot, *R. n.*, 1905.

at the close of the second or third decade, seldom later. Women are somewhat more liable to it than men.

Heredity is the most important factor in the etiology. Many other exciting causes, such as mental exhaustion, prolonged emotion, work in over-heated rooms, and possibly also onanism may produce migraine in predisposed individuals. It may undoubtedly have a reflex origin. Some observations point to the fact that diseases of the mucous membrane of the nose (proliferation of the mucous membrane, enlargement of the erectile tissue on the turbinated bones) are specially apt to have this effect. The connection with affections of the sexual system, entozoa, etc., is less definite, and the relation of migraine to gout is not sufficiently explained, although it has been emphasised by Gowers and Charcot.

The individual attacks may be brought on specially by excitement, alcoholic excess, mental exhaustion, bad air, and sometimes by coitus (Determann). Markus blames variations in the atmospheric pressure.

The most important and often the only symptom of the disease is *a periodic attack of violent headache*, which is associated as a rule with *gastric disturbances*, loss of appetite, nausea, flatulence, and vomiting. The attack lasts for twelve to twenty-four hours, but it may pass off in two to three hours or persist for two and even three days.

The headache does not as a rule commence in its full intensity ; it is usually preceded by a feeling of exhaustion, drowsiness, a tendency to yawning, fulness in the head, giddiness, depression, etc. One of my patients suffered on the evening before the attack from bulimia, another from thirst, and a third from great excitement. The headache is at first usually dull and comparatively slight ; it gradually increases, often to a degree which may be called unbearable. It is by no means always limited to one side of the head—as one would conclude from the name *hemicrania* —although it is often confined to the left side, but it frequently affects the whole of the frontal region, the forehead, or the temples of both sides, especially the parts round the eyes, or is felt most intensely at one spot and then at another. It may also affect the occipital region. In one of my cases it was localised in the region of the root of the nose. It occasionally begins on one side and then passes over to the other ; the patient sometimes states definitely that the site of the pain alternates regularly in each successive attack.

In some rare cases there is hyperæsthesia of the scalp and tenderness of the emerging branches of the fifth nerve to pressure during the attack. I have more frequently found tenderness on pressure upon the superior cervical ganglion of the sympathetic during and sometimes apart from the attacks.

During the attack of pain the patient feels exhausted and wretched and is abnormally sensitive to sensory impressions. He can neither bear bright light nor noise. In order to avoid these he darkens his room and shuts himself off as far as possible from the outer world. Movement of the head or eyes intensifies the pain. The appetite is usually completely lost. If *vomiting* is present, it may occur at the height of the attack or towards its close. Diarrhœa and polyuria occasionally appear towards the end of the attack. Atony and dilatation of the stomach is, according to Mangelsdorf,[1] a common accessory or resulting symptom of migraine.

[1] *B. k. W.*, 1903.

Salivation rarely occurs. Secretion of tears and sweating (Liveing) are unusual symptoms. Pain in the region of the kidneys and albuminuria have also been described, but it is doubtful whether this so-called renal migraine (Sticker, Markwald) has anything to do with hemicrania. Conjunctival and retinal hæmorrhage (Brasch-Levinsohn [1]) and nasal hæmorrhage (Rossolimo) have been observed.

The *crisis* usually ends in sleep ; the patient wakens without any pain in his head and feels quite well. If the attacks are severe, coughing, straining, sneezing, etc., may give rise to transient pain in the head for one or two days after the attack is over. Slight attacks are sometimes arrested by occupation, eating (Möbius), etc.

The attacks are separated by intervals of varying length. As a rule several weeks intervene, but the attacks may come on only every second month, or as often as several times in the week. The intervals are not usually of the same duration, even in the same individual, but the migraine may return *regularly every three or four weeks*, especially in women, when they bear some relation to menstruation.

Vasomotor and pupillary symptoms are among the inconstant phenomena of migraine, to which Du Bois-Reymond [2] and Möllendorf [3] have drawn attention. It is assumed that they correspond to a condition of irritation or paralysis of the sympathetic. Thus in one case the face became pale, the skin was cold, the temporal artery contracted and hard, the pupils dilated, and the secretion of saliva increased ; in the other the face and conjunctivæ became red, the arteries dilated, and the pupils contracted. Unilateral hyperidrosis has sometimes occurred. These symptoms have led to the differentiation of two forms of hemicrania : *hemicrania sympathico-tonica* or *angio-spastica*, and *hemicrania sympathico-paralytica*. All these signs are very seldom present in one case ; the symptoms of irritation and paralysis may blend together or may both be present at the same time and on both sides ; and, finally, there is a large number of cases in which no abnormality can be detected, either in the pupils or the colour of the face. At all events the few existing cases of this kind do not justify us in attributing hemicrania to an affection of the sympathetic.

But there is a class of cases in which the attacks of migraine are associated with *functional disturbances of the nerves of special sense, of sensibility and motility*, and even by *disturbances of speech*.

Ocular migraine (hemicrania ophthalmica, migraine ophthalmique) is the most common form. The attack is ushered in by shimmering before the eyes, usually accompanied by a disturbance of sight. A bright spot appears at some point of the field of vision, which spreads or takes the form of a shining zigzag figure (Fig. 413) ; this gradually fills the whole field of vision with dazzling, sparkling, blinding light, sometimes in bright shades of colour. The visual disturbance which comes on at the same time has the character of *scotoma* (scintillating scotoma), of partial or complete *hemianopsia*, or less often of *amaurosis*. All these symptoms, which may vary in numerous ways, only last for a short time (a few minutes to half an hour) before the headache commences. These optic phenomena have been fully described by Charcot [4] and Jolly.[5]

[1] *B. k. W.*, 1898. [2] *A. f. Anat. u. Phys.*, 1860. [3] *V. A.*, Bd. xci.

[4] "Clinique des maladies du syst. nerv.," 1889-91, and "Leçons du mardi."

[5] *B. k. W.*, 1902. See also Féré, *R. d. Méd.*, 1881 ; Gowers, *Trans. Ophth. Soc. London*, 1895 ; Manz, "Über Flimmerskotom," *N. C.*, 1893 ; Sigrist, "Mitt. aus d. Klinik. der Schweiz.," i., 1894, etc.

Hoeflmayr (*N. C.*, 1903) describes unusually long duration of the scintillating scotoma in an otherwise atypical attack of migraine.

There are only a few cases in which the patient lost his power of speech and was unable to form words during the attack. This *aphasia* is usually incomplete, or, as in a case under my observation, it may be associated with agraphia. Word-deafness has only been noted in rare cases. It lasts for a few minutes to a quarter of an hour, and is followed by pain, always in the *left* side of the head. Hemianopsia and aphasia may occur simultaneously.

I have treated a man, who along with three other members of his family had suffered from childhood from hemicrania ophthalmica with aphasia. There was no hereditary nervous taint, but the father had used a great deal of potassium cyanide in the course of his work.

Paræsthesiæ in one arm, or one or both sides of the body (*e.g.* the lips, tongue, etc.), are frequent accessory symptoms of the attack, but a corresponding anæsthesia is not often present. A feeling of *weakness* in one arm or one side of the body, sometimes accompanied by true paresis, may be one of the localising symptoms of migraine. If it affects the limbs of the right side, the headache is localised in the left side of the head.

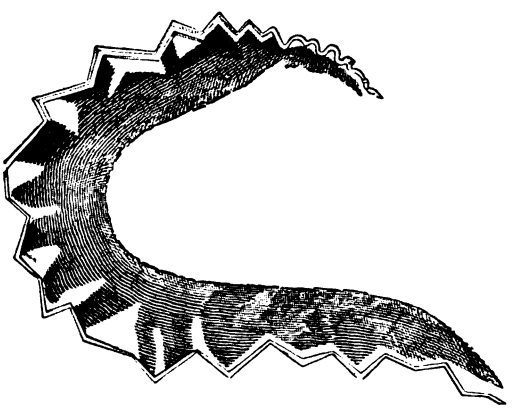

FIG. 413.—Representation of scintillating scotoma. (After Charcot.)

In a single case under my observation each attack of migraine commenced with typical cerebellar symptoms. During the attack standing and walking were very uncertain; the patient swayed like a drunken man, had severe vertigo, and a feeling as if his body or some portions of it were doubled. The impairment of balance appeared and disappeared with each attack. The case might have been called one of *hemicrania cerebellaris*.

Hemicrania may also be associated with mental disturbances (Griesinger, Krafft-Ebing,[1] Mingazzini[2]), usually in the form of transitory insanity, a condition of excitability, confusion, or stupor associated often with hallucinations, especially of the visual type, which rapidly pass off. Mingazzini gives the term dysphrenia hemicrania transitoria to this psychosis.

The symptoms just described may be combined in various ways. They are sometimes repeated in a stereotyped way in each attack, but are more often extremely irregular; they may be fully developed during one paroxysm and entirely absent or very incomplete in another. It is not unusual for a person who has suffered for a long time from simple migraine to become subject at a later period to these complicated attacks.

[1] *W. kl. R.*, 1895, and *Arb. aus d. Gesamtgeb. d. Psych. und Neurol.*, 1897 and 1899.
[2] *Riv. di Fren.*, 1893, xcv., and *M. f. P.*, i. See also Consiglio, *Policlinico*, 1905.

With the exception of the paroxysms, there are no objective signs of disease. Migraine is, however, so often associated with *neurasthenia* and *hysteria* that signs of these neuroses or troubles of a similar kind may be present during the intervals. General nervousness almost always develops when the condition has persisted for a long time and the attacks have been very frequent and severe. The patient usually bears a look of suffering and becomes prematurely grey and aged.

Hemicrania also tends to be associated with other diseases of the nervous system, such as *writers' cramp*, *tic convulsif*, etc. Its relations to *epilepsy*, which have been specially studied by Liveing, Gowers,[1] and Möbius, with whose opinions Cornu [2] and others agree, are of special interest. A person who has suffered for a long time from migraine may become subject to epilepsy. Thus I have treated a man for severe congenital neurasthenia and hemicrania, in whom at the age of thirteen the paroxysms of hemicrania were replaced by true spasmodic attacks with loss of consciousness and biting of the tongue; when he reached the age of seventeen the migraine again reappeared in place of these attacks. A woman who had suffered from childhood from hemicrania with vomiting, etc., found the condition greatly intensified after an abdominal operation, and at the height of the migraine, or alternating with it, she suffered from deep unconsciousness with passage of urine and fæces. But apart from these combinations, there can be no doubt of the *relationship* between the two neuroses. In both we have the paroxysmal occurrence of the nervous symptoms, the same prodromata, and the termination of the attack in sleep. *Hemicranial equivalents* corresponding to the equivalents of the epileptic attack have also been observed (Möbius). A *cardialgia*, a condition of violent giddiness or an attack of convulsions may to some extent vicariously take the place of an attack of migraine. Nevertheless there is no justification for regarding hemicrania as a variety of epilepsy, and Strohmayer [3] in particular has recently protested against the assumption of a close relationship between these different affections. It has never been proved that one of these conditions may develop into the other. Möbius regards gastroxynsis as a variety of migraine. On the other hand attacks which merely resemble those of hemicrania may be brought on by hyperacidity of the gastric juice (Fenwick). I have seen cases in which true hemicrania was replaced by *violent pain* at some circumscribed part of the trunk or an extremity, which after lasting for some hours or even for a whole day disappeared spontaneously, returning in the same way a few weeks later. Lamaq has confirmed this observation. In another case attacks of *hemiparesis* alternated with the migraine.

It should be noted that *paralysis of the ocular muscles* may occur in migraine; so-called *periodic oculo-motor paralysis* is apparently closely related to this condition (see, however, p. 473). I have several times found persistent paresis of one or more ocular muscles (abducens paresis, ptosis, ophthalmoplegia interna: Troemner found the latter in one case) in persons who suffered from migraine, but I could not definitely discover whether this was an unusual symptom of the migraine or the prodroma of an organic disease of the brain. Karplus (*Jahr. f. P.*, 1902) describes paralysis of an ocular muscle which only occurred during an attack of migraine. I have sometimes found permanent contraction of the palpebral aperture and pupil of the affected side in persons subject only to unilateral migraine (see below). Féré (*Rev. de Méd.*, 1897) has reported

[1] See his latest contribution, "The Borderland of Epilepsy: Migraine," *Brit. Med. Journ.*, 1906.

[2] *Thèse de Lyon.* See also Epstein, *Budap. Orv.*, 1904; Kowalewski, *Arch. de Neurol.*, xx.

[3] *M. m. W.*, 1903; *M. f. P.*, xiii.

spastic symptoms in the eye muscles, especially the orbicularis oculi, during an attack of migraine, and for this reason he speaks of a " migraine ophthalmo-spasmodique."

Hatschek and Rossolimo (*N. C.*, 1901) also relate a form of recurring facial paralysis to migraine, but this connection is by no means clear.

I have once seen an optic neuritis with paracentral scotoma develop in a patient who suffered from hemicrania, vasomotor coryza, and other vasomotor disturbances. I record this case without drawing further conclusions from it, as it is unique. Alternating attacks of hemicrania and angioneurotic œdema have been mentioned by Otto (*Pet. med. W.*, 1906).

It is doubtful whether *mental disturbances* occur as equivalents of hemicrania, although such disorders appearing in those suffering from migraine and apparently replacing the paroxysms of pain have been interpreted in this way. These disorders took the form of attacks of mania (Féré), acute confusion, and in one of my cases of melancholia.

I have occasionally found in hysterical persons suffering from migraine, that *hemianæsthesia* associated with sensory disturbances was present on the corresponding side.

Another form of migraine is characterised by the *constancy of the headache*. It might be termed *hemicrania permanens*. Féré [1] speaks of an *état de mal migraineux*, and Möbius of a *status hemicranicus*. It is to be recognised from the fact that it occurs in individuals who have for a long time suffered from typical attacks, or whose parents had true migraine. I have noticed the transformation of paroxysmal migraine into the *chronic form* specially in *neurasthenic* or *hysterical* patients.

On the other hand there are incompletely developed attacks, which represent an abortive form of true migraine, and consist merely of shimmering before the eyes of short duration, very transient nausea, with slight fulness in the head, etc.

Pathology.—It should not be assumed that the condition is due to an organic disease. There is some evidence pointing to a *vasomotor* origin of the attacks. This evidence rests not so much upon the vasomotor disturbances which are sometimes present and which may be the effect of the pain, as upon the *focal cerebral symptoms* described above, the transient nature of which points to temporary disturbance of the nutrition of the part, which could best be explained as due to a *vascular spasm*. Less objection can be raised to this view (apart from the uncertainty which still reigns with regard to the vasomotor innervation of the cerebral vessels), as these cerebral focal symptoms usually precede the attack of pain and cannot therefore be a result of it. A case of Jacobsohn's,[2] in which the hemicrania was associated with signs of paralysis of the sympathetic (and exophthalmic goitre) gives us further reason to suspect a close connection between migraine and the vasomotor system.

I have observed the following case: a woman, who since her childhood had suffered from severe left-sided hemicrania, showed persistent contraction of the left pupil and palpebral aperture, and she had never perspired in her life. (Her son is imbecile, suffers from hemicrania and epilepsy, and has six toes on the left foot.) This origin of the disease is also supported by the sensitiveness of the superior sympathetic ganglion which I have found in many cases.

Many theories have been advanced to explain migraine. It has been attributed to anomalies of metabolism and consequent intoxication. One writer states that he has found toxic products in the urine, but it seems to me very doubtful whether these should be regarded as the cause of the attacks. Haig thinks they are due to excess of uric acid in the blood. Decomposition of the contents of the stomach, anomalies of refraction, diseases of the naso-pharynx, etc., have all been regarded as causes of migraine.

[1] *Rev. de Méd.*, 1892.

[2] *D. m. W.*, 1898.

Spitzer ("Über Migräne," Jena, 1901) has suggested that it may be caused by abnormal narrowness of the foramen of Monro; should the brain become affected by hyperæmia involving the choroid plexus, the result would be complete displacement of the foramen of Monro, occlusion of the ventricular fluid, distension of the whole hemisphere, and compression against the skull-cap, etc. Lévi (*R. n.*, 1905) thinks the disease arises from the medulla oblongata.

The theory of vascular spasm throws light also upon the fact that the transient paralytic symptoms of complicated attacks of migraine, viz. the hemianopsia, aphasia, etc., may become chronic (Charcot, Oppenheim). I [1] have shown in one such case that the paralytic symptoms were due to *thrombosis of the internal carotid* (shortly before the emergence of the artery of the Sylvian fissure). Infeld has reported a similar case, and that of Thomas,[2] in which a permanent hemianopsia developed in the course of the migraine, should also be included here.

Diagnosis.—In typical cases the condition can never be mistaken. Headache caused by cerebral tumour, aneurism of the cerebral arteries or uræmia, and associated with vomiting, may be in rare cases confused with migraine, but this error may always be avoided by careful examination. Although in cerebral tumour the pain may come on in paroxysms during the initial stages, other signs are sure to be present which confirm the diagnosis. In any case, it should be remembered that slowing of the pulse has been observed at the height of an attack of migraine ; in one of my cases it slowed down to forty-eight beats a minute. If the hemicrania commences in early childhood, it may be difficult to diagnose. I have seen cases of this kind which had been diagnosed as cerebral tumour or meningitis. But the condition can usually be quickly recognised from the periodicity of the headache, the similar duration of the various attacks, and the patient's satisfactory general condition and freedom from objective symptoms between the attacks. Evidence that the parents have also suffered from hemicrania will strengthen the diagnosis.

Course and Prognosis.—The disease almost always lasts for the greater part or even the whole of the patient's lifetime, but it may disappear at the climacteric or later. More rarely it yields spontaneously or to treatment at an early period of life. Pregnancy, the onset of menstruation, or change to another climate may effect a cure. In a few of my cases the hemicrania disappeared after an injury to the head. In one patient who had suffered from it since youth, it was absent for twelve years after typhoid fever. One man who consulted me for neurasthenia had had severe migraine from childhood to the age of thirty, when it entirely disappeared. His physician had emphatically assured him that it would vanish during his thirtieth year. In another case the migraine persisted between the ages of eight and fifteen, and then gradually disappeared, giving place to severe and incurable neurasthenia. In not a few cases the vomiting which accompanied the pain has ceased at a later period, and in one of my patients the hemiopia associated with the earlier attacks disappeared.

Hemicrania sometimes, though very rarely, develops into epilepsy, but in most cases the attacks are usually of the so-called intermediary (Oppenheim) or psychasthenic nature (see p. 1135 and the footnote to p. 1135). It has also been observed that migraine forms a premonitory symptom of *tabes dorsalis and paralytic dementia*. It has disappeared as the tabes developed, or the attacks have taken the form of gastric crises. As to the relation of migraine to paralytic dementia, the form associated

[1] *Charité-Annalen.*, xv. [2] *Journ. Nerv. and Ment. Dis.*, 1907.

with symptoms of loss of brain function gives occasion for a certain amount of anxiety, as in some very rare cases it may develop into general paralysis. Karplus [1] emphasises the great variability and irregularity of the single attacks of this symptomatic form of migraine.

The *prognosis* of hemicrania as to recovery is therefore not on the whole very hopeful. On the other hand, life is hardly in any danger from the disease, apart from the very rare cases in which the vascular spasm leads to thrombosis. The prognosis is rendered somewhat graver by the fact that migraine has a tendency to be combined with hysteria, neurasthenia, or less often with epilepsy, etc., and, in rare cases, to develop into tabes dorsalis or general paralysis. Ophthalmic migraine is more serious in this respect than the other forms, if it appears first in old age. On the whole, however, we may regard even this form as without danger. The hysterical form of hemicrania has a much more favourable prognosis than the true form, and in view of the frequency of the combination of these conditions, there is always some suspicion that hysterical attacks may occur even in true migraine.

Treatment.—The factors which bring on the paroxysms of migraine should in every case be ascertained, and taken into consideration as far as possible with regard to the treatment. Advice as to the mode of life and the diet may be of more benefit than drugs. The patient usually learns for himself to avoid the causes which excite the attacks.

In many cases the patient suffers from chronic *constipation*, and regulation of the bowels may have a good effect. Thus excellent results have followed the use of *Carlsbad water* or similar saline, and *cold-water enemata*. The condition has been improved and even cured in some of my cases by treatment of a *chronic nasal affection*; in one case the attacks ceased after the removal of hypertrophied tonsils. Hartmann (*D. m. W.*, 1907) asserts that he has repeatedly succeeded in curing migraine in children by the removal of adenoids. If anæmia is present, *preparations of iron* have sometimes a good effect. On the theory that the attacks may be due to auto-intoxication, baths in a vapour box (Steckel), and washing out the stomach (Frieser, Aikin) have been recommended in addition to dietetic measures (vegetable diet, alkaline water, etc.).

A change of air, and especially a prolonged residence in the hills or at the seaside, may have an excellent effect.

In addition to these methods for the cure or improvement of the disease, *cold-water treatment*, *general massage*, *gymnastics*, *general faradisation or Franklinisation*, *galvanic treatment* of the brain, sympathetic, etc., have been prescribed. They should certainly be tried in every case, but the result is on the whole very disappointing. Counter-irritation is seldom of use, but I have occasionally seen excellent results from the *application of a seton*; this treatment has recently been successful in one desperate case, and Whitehead reports a similar result.

Drugs may also be given. *Arsenic* is specially good, and its use has led to improvement in many of my cases and to recovery in one. In another case the attacks were so severe that the patient entertained the idea of suicide, and every method failed until I sent him for a course of strong arsenical treatment at Levico, after which the attacks ceased for a number of years. He had been so reduced by the migraine that he gained 10 lbs.

[1] *W. kl. R.*, 1903.

in weight during the first four months in which he was free from attacks of it. The drug is best given in the form of *arsenious* acid, possibly combined with iron. The waters of Levico, Roncegno, and Guber may also be prescribed in corresponding doses. Recently cacodylate of soda, atoxyl, arsenferratose, and other preparations of arsenic have become known. The continuous use of *bromides* has been specially recommended by Charcot. Möbius thinks this drug should be used in increasing doses in severe cases. If bromide cannot be borne, he prescribes salicylic acid.

Sarbo advocates long-continued use of iodide of potassium and bromide of potassium, in a dose of 0·5 (7·5 grs.) once a day. Mendel's prescription runs: sodium bromide 2·5 (40 grs.), sodium salicyl. 0·25 (4 grs.), aconitin 0·0001 ($\frac{1}{600}$ gr.) in powder. One powder every morning to a cup of valerian tea, for twenty days; to be resumed after an interval of ten days.

For the angiospastic form Gowers recommends nitroglycerin in doses of from 0·00025 to 0·0005 ($\frac{1}{2400}$ to $\frac{1}{1200}$ gr.) three times a day, in the form of tablets, or better still in alcoholic solution (1 per cent.). The dose of this is one drop with the addition of dil. hydrochloric acid or tinct. nux. vom., etc. Nitrite of sodium (30 grains in 503 of water, a teaspoonful several times a day) and adrenalin in a dose of $\frac{1}{4}$ milligramm ($\frac{1}{2600}$ gr.) (Kreuzfuchs) may also have some effect.

Most of the drugs used in migraine serve only to *mitigate each individual attack*. A number of drugs may shorten an attack and diminish the intensity of the pain or even relieve it completely. But every drug has not the same effect upon every individual, and each in time loses its effect.

In many cases *sodium salicylate* (2·0 to 3·0 (30 to 45 grs.)) in water or, better still, in a cup of black coffee, soothes the pain. *Antipyrin*[1] (0·4 to 1·0 (6 to 15 grs.)) and sometimes *phenacetin* (0·75 to 1·0 (12 to 15 grs.)), *caffeine citrate* (0·15 (2·5 grs.)) or *caffeine and sodium salicylate* (0·2(3 grs.)) may also do good, and in a few cases *antifebrin* (0·25 to 0·3 (3·5 to 5 grs.)), *exalgin* (0·25 (3·5)), *analgen* (0·5 to 1·0 (7·5 to 15 grs.)), lactophenin, pyramidon (0·3 to 0·5 (5 to 8 grs.)), cryophen (0·5 to 1·0 (5 to 15 grs.)), aspirin (1·0 to 2·0 (15 to 30 grs.)), euchinin (0·1 to 0·2 (1·5 to 3 grs.)), *methylene blue* (0·1 ($1\frac{1}{2}$ grs.)), trigemin (0·5 to 1·0 (8 to 15 grs.)), validol (5 to 15 drops). Pasta guarana or paullinia sorbilis (2·0 to 4·0 (30 to 60 grs.)) and amylnitrite are recommended (in the spastic forms), a few drops on a handkerchief being given to inhale. Diuretin, and in the paralytic form *ergotin* (extract of ergot aq., 2·5 (4 grs.)), spirit. dilut. and glycerine, aa 5 (7·5 grs.), one-sixth being used to a syringeful, *cytisin* (0·0003 to 0·0004 ($\frac{1}{2000}$ to $\frac{1}{1500}$ gr.)), and *migranin* (1·1 (17 grs.), for a dose), may also be prescribed. I cannot say that migranin deserves its name, and in a few cases the use of the doses prescribed was followed by symptoms of poisoning which were attributed to the antipyrin which it contains. Liégois recommends the combination of sulphonal (1.0 (15 grs.)) with phenacetin (0·5 (7·5 grs.)), and Strauss advocates migraine tablets composed of phenacetin (0·5 (7·5 grs.)), caffeine (0·06 (1 gr.)), codein (0·02 ($\frac{1}{3}$ gr.)), and guarana (0·2 (3 grs.)). I have found potassium bromide in combination with caffeine of value in some cases.

A cold compress or firm bandage round the head, or menthol (in alcoholic

[1] This drug is generally found by the patients to do good, but I have occasionally seen a paradoxical effect, as it intensified the pain and increased the number of the attacks. The toxic effects which occasionally occur are well known, especially the exanthema. The dose of phenacetin and lactophenin requires to be carefully considered.

solution 3 to 20), or in the form of a cone rubbed upon the forehead or temples, or cocain dropped into the nostrils (Dobisch) may have a soothing effect. A hot foot-bath or a mustard plaster to the nape of the neck may also be helpful. In some cases hot compresses or packs to the head alleviate the pain. One woman stated that staying in the kitchen or beside a hot fire greatly relieved the pain. In a few cases cocaine dropped into the conjunctival sac had a good effect. Subcutaneous injections of morphia are very seldom necessary, but I have known a few cases of such extreme severity that they could only be controlled by morphia, and even this failed after being almost constantly used.

The application of electricity during the attack in the form of the electric hand, etc., or galvanisation of the sympathetic, has usually no effect, but it may do very well in the hysterial form. The attack has occasionally been arrested by the spark-discharge of the Franklin current (Determann). In addition to the congenital and hysterical forms of migraine, there are some cases in which infrequent attacks of true migraine are associated with attacks of psychogenic headache of a similar character, and in these hypnotism may be of use.

Many patients refuse all treatment; they remain quietly in a dark room, eating nothing or taking only a little tea, seltzer, lemon juice, etc., and endure the pain with patience.

Headache (Cephalalgia, Cephalæa [1])

Headache is a symptom of various kinds of diseases of the nervous system and other organs. It is seldom an *independent*, primary disease.

The headache which accompanies organic brain diseases need be considered here only in so far as it requires to be discussed from the diagnostic point of view. Hemicrania has been considered in the previous chapter.

Headache is often caused by *circulatory disturbances* within the cranial cavity. It may be due both to hyperæmia and to anæmia, and more especially to variations of the blood-pressure in the brain. *Active hyperæmia* gives rise as a rule to violent, throbbing, sometimes pulsating pain in the head, often associated with vertigo, noises in the ears, shimmering before the eyes, flushing of the face, conjunctiva, etc. There are some cases in which this condition is due to *primary vasomotor disturbances*. These flushings come on from time to time; the face and ears become red, the skin feels hot, the pulse is full and rapid, etc. This form of headache is known as *cephalalgia vasomotoria*. It may be combined with other vasomotor disorders (urticaria, etc.). Each attack is brought on by mental overstrain, emotional excitement, alcoholism, excessive smoking, onanism, or injury to the head. In a few of my patients spots of redness in the face and temples appeared during the attack, the pain being localised to these spots. *Venous hyperæmia* of the brain is an important cause of chronic headache, especially when it is of marked degree, as in diseases of the heart, pulmonary emphysema, etc. Severe, persistent coughing, and compression of the veins of the neck by a narrow collar may have the same effect.

Anæmia, whether chlorotic in nature or caused by loss of blood, is

[1] Earlier literature in Erb, *Ziemssen's Handbuch*; also in Möbius, "Der Kopfschmerz," Halle, 1902. Otherwise dispersed.

almost always accompanied by headache, which is usually dull and heavy, seldom violent, and is situated either in the forehead and eyes or in the temples, and sometimes over the whole head. The headache caused by hyperæmia is intensified by bending, coughing, straining, sneezing, etc.; that due to anæmia is usually relieved by the recumbent position.

The headache which so often accompanies *arterio-sclerosis* is chiefly caused by *disturbances of the circulation and nutrition.* It is possible, however, that the rigid walls of the vessels may produce a chronic condition of irritation in the surrounding meninges which finds its expression in headache.

In a few cases the trouble is of *toxic* origin, occurring *e.g.* in acute alcoholic intoxication (circulatory disturbances may possibly also be concerned), *nicotin*, *caffeine*, *morphia*, *chloroform*, *ether*, and *metallic* poisoning.

The headache caused by *gastric disorders* (indigestion, catarrh of the stomach, *constipation*), which is one of the most common forms of this trouble, is possibly due chiefly to the presence of toxic products in the blood, as has been again recently emphasised by Lauder-Brunton. It may, however, be partly of reflex origin. The headache which occurs with intestinal parasites, *disorders of menstruation*, etc., points to this mode of origin. Headache accompanied by fever is also chiefly due to poison. *Autointoxication* is the cause of *urœmic*, *acetonic*, *diabetic* headache, etc. Infective diseases (typhoid, influenza, malaria) may be followed by persistent headache. *Rheumatism* of the muscles of the neck (frontalis, occipitalis, etc.) causes a boring, tearing pain, which is intensified by pressure on the muscles and movement of the scalp. Nodules and swellings are in such cases sometimes found in the subcutaneous tissue of the neck and occiput, and these may be very painful on pressure (Edinger,[1] Auerbach, Norström [2]). According to Rosenbach [3] and Peritz,[4] the muscles or their insertions are usually tender to pressure, and there may also be hyperæsthesia of the skin above the muscles.

Diseases of the *cavities* adjacent to the skull (nasal, frontal, pharyngeal, tympanic, etc.), and of the *mucous membrane which lines them*, may be the source of the pain. Bresgen [5] and Hartmann [6] have pointed out the frequency of these relations. Carious teeth may give rise to headache, usually localised in the temporal region. *Anomalies of refraction*, especially hypermetropia and disorders of accommodation, are additional causes, but the importance of these factors is over-estimated by some physicians. Walton [7] has recently strongly emphasised this connection. There are cases in which any eye-strain is followed by headache. Sexual excess, especially masturbation, may also be considered a cause.

Insomnia, *mental exhaustion*, working in over-heated rooms, or even near a gas jet or a lamp, and above all *trauma* may be causes. Injuries which do not directly involve the brain and its membranes may also result in headache (especially the vasomotor form).

In the great majority of cases the headache is due to *neurasthenia*, *hysteria*, and *hypochondria*. Even where it is apparently the only symptom, or the only trouble of which the patient complains, it is often a product of these conditions. It should not be forgotten that the symptoms of these neuroses may disappear for a considerable time, giving

[1] *Deutsche Klinik*, etc., Bd. iv.
[2] "Chronischer Kopfschmerz," etc., German ed., 1903.
[3] *D. m. W.*, 1886.
[4] *Med. Klinik*, 1906.
[5] *M. m. W.*, 1893.
[6] *D. m. W.*, 1907.
[7] "Contrib. for the Mass. Gen. Hosp.," Boston, 1906.

place to one single disorder. Careful investigation will reveal the neurasthenic or hysterical nature of the pain. Some cases remain, however, in which there is no question of a general neurosis, and in which the *headache in itself represents the disease*, but the factor of *heredity* often enters even into such cases. It has already been mentioned that hemicrania may lead to *chronic* headache, and it may also be the cause of the appearance of other forms of headache in the descendants. Charcot speaks of a cephalæa adolescentium as an independent disease.

The peculiar characteristics of the headache of hysteria, neurasthenia, and migraine have been described under these headings. *Chronic headache* may come on periodically, but not at regular intervals, and the attacks vary greatly in their duration. In some cases the pain is described as constant, with only occasional remissions. If it is violent and persistent, it affects the general condition, gives rise to depression, makes the patient appear ill and prematurely aged, and leads him to become hypochondriacal.

In a few very severe and obstinate cases under my care the headache was always brought on by sleep. The patient wakened during the night or in the early morning with violent headache, which disappeared in the course of the forenoon. If he did not sleep during the night, or kept intentionally awake, the pain as a rule did not come on.

I have found affections of the cervical sympathetic associated with headache which affects the whole side of the head with the exception of the mucous membrane, or certain parts of this region, and which may be associated with symptoms of irritation or paralysis in the nerves. The pain in such cases is often in the neck, larynx, and pharynx.

The *prognosis* depends upon the primary cause. Chronic primary headache is not infrequently an *intractable* disease which persists for years or even for the whole life.

Recognition of the cause is the most important factor in the *treatment*. One should never be satisfied with merely making a diagnosis of headache. A careful examination of the whole physical condition and a minute consideration of all the factors which might be concerned is the only means of achieving improvement or recovery. Headache, especially when persistent and violent, indicates the necessity for ascertaining if any symptoms of a brain disease (brain tumour, syphilis, meningitis, etc.) are present. The skull, cranial cavities, and special senses should also be examined. It need hardly be said that cicatrices, depressions of the bone, etc., at the site of the pain should be surgically treated. Errors of refraction should also be corrected. The condition of the heart, the vessels, and the urine may afford important indications. The functions of the stomach should be tested with the proper methods. It seems to me that in many cases a thorough *investigation* is neglected ; this should bear not only upon the personal condition of the patient, but also upon his occupation, his mode of living and working, and the circumstances of his heredity.

This method will lead to rational treatment.

We cannot here show in detail how dietetic treatment will be suitable for one case, *aperients* or remedies for tape-worms in another, *physical exercise* in a third, *blood-letting* or *counter-irritants* in a fourth. If anæmia is present, preparations of iron, which may perhaps be replaced by small doses of arsenic, have often a good effect. Some cases are known in which

replacement of a retroflexed uterus has immediately cured the headache (Olshausen). Correction of errors of refraction has sometimes an excellent effect. *Hydrotherapeutic* treatment, viz., cold rubbings, lukewarm half baths, warm or cold foot-baths of short duration, hot douches or wet packs to the feet, etc., are often of use in *congestive cephalalgia.* Hot full baths have also been recommended, *e.g.* for the congestive troubles of the climacteric (Gottschalk). These measures may also relieve and cure *neurasthenic headache.* For this and similar forms electricity, applied in various ways, Franklinisation in particular, may be prescribed, and a change to the seaside, the hills, or simply a change of air, a *sea-voyage,* or a journey to the *south* may in many cases be successful. *Psychotherapy,* it need hardly be said, may be used with good results.

Many simple remedies, such as a cold compress, rubbing with menthol, an ether spray (or the application of ethyl-chloride), massage to the nape of the neck, hard brushing of the scalp, inhalation of sal volatile, local application of chloroform, etc., are well known to the patient himself, and may be useful in the mild forms. Hot compresses to the head are sometimes soothing. One of my patients stated that his headache disappeared after driving in a carriage over a bad road or after a railway journey; the vibration massage which I prescribed for him had also an excellent effect. Nägeli's method may be used with advantage in a few cases. Rheumatism of the muscles of the head will be suitably treated by diaphoresis, massage, and local faradisation. Schleich's method of infiltration anæsthesia—intracutaneous injection of a solution of 0·2 per cent. each of tropacocain and sodium chloride—may do good (Bloch), and so may injection of about 0·5 c.cm. of a salt solution of 0·2 : per cent. into each painful point. This is warmly advocated by Peritz.

As regards the *drugs* that may be used, the reader should refer to the preceding chapter. Almost every one of the drugs there mentioned has been recommended for the other forms of headache, occasionally with good results. Of the newer drugs we may mention saloquinine (15 gr.) anæsthesin (3 gr.), trigemin (7½-15 gr.) (a combination of pyramidon and blutylchloral), and nervol (vannadium citrate with bromlithium, in teaspoonful doses). I have had no satisfactory results from methylene blue, which has been recommended by others, but have found pyramidon very useful. Diuretin (15 gr. thrice daily) has often proved very good in the headache of arterio-sclerosis. One should be very careful and restrained in the use of narcotics.

There are in addition a number of very stubborn cases in which none of these remedies are really of much use. In such cases persistent derivation by the application of a *seton* or the *button cautery* to the neck has sometimes proved successful. In a few cases, I have seen a headache which had persisted for years, and deprived the patient of his power to work, entirely disappear after the use of a seton (see, however, the remarks on p. 286).

Vertigo

Literature in Nothnagel, Ziemssen's "Handbuch," xii.; Mach, "Grundlinien der Lehre von den Bewegungsempfindungen," Leipzig, 1875; Hitzig, "Der Schwindel," Nothnagel's "Handbuch," xii. 2; Wanner, "Über die Ersch. von Nystagmus bei Normalhörenden, Labyrinthlosen," etc., München, 1901; Panse, "Der Schwindel," *Z. f. Ohr.*, 1902; Bonnier, "Le Vertige," second edition, Paris, 1905; Stein, "Über Gleichgewichtsstör. bei Ohrenleiden," *Z. f. Ohr.*, Bd. xxvii.; Frankl-Hochwart, "Der Ménièresche Symptomenkomplex," second edition, Wien, 1906 (Nothnagel's "Handbuch"); *ibid.*, *Jahrb. f. P.*, 1905; Bárány, "Unters. über die V. Vestib. des Ohres refl. ausgel. Nyst.," etc., Berlin, 1906 (with bibliography); Passow, *B. k. W.*, v., and "Die Verletzungen des Gehörorgans," Wiesbaden, 1905; Gowers, "Vertigo," etc., *Brit. Med. Journ.*, 1906.

Vertigo is a symptom of varying importance, as diseases of very different character may give rise to it, and in a very few instances it may be an independent affection. The fact that it may be the *only* or the most important symptom of a morbid state justifies us in discussing it separately.

By the term vertigo we understand a *feeling of discomfort arising from a disturbance of the relations of our body to space*, or a feeling of loss of balance. The organs which serve for the maintenance of equilibrium—the cerebellum, labyrinth, and the nerve tracts which connect these with each other and with the cerebrum—have the greatest influence in bringing about this sensation (see pp. 643 *et seq.* and p. 1043).

We need not here discuss the cases observed after injury to the semicircular canals, nor the theories of Flourens, Goltz, Ewald, Cyon, Mach, and Breuer, and the objections raised to them by other writers. We shall only point out that Breuer thinks both the semicircular canals and the otoliths play a part in the sensations and the regulations caused by changes in the position of the head and body. The former are said to become active in forward movements, the latter in the movement of rotation.

We do not definitely know how it happens that lesions and diseases of the parts concerned in the maintenance of equilibrium sometimes produce a feeling of giddiness or only loss of balance, and sometimes both these disturbances. The way in which the giddiness comes on is apparently of importance in this respect, as an *acute* onset is usually accompanied by giddiness, which is more often absent if the development is slow and not associated with marked variations in the intensity of the process. We can also imagine that morbid processes which affect not only the parts concerned in maintaining equilibrium and the tracts leading to them, but also interrupt those connecting them with the cerebrum, may be unaccompanied with any feeling of giddiness. Affections of the cerebrum itself—probably of the areas connected with the cerebellum, viz., the frontal lobes and possibly the central area—may naturally be accompanied by sensations of giddiness. Lesions of the labyrinth are specially apt to produce vertigo, as here the structures which play so essential a part in preserving the balance are compressed and confined within a narrow area. These structures are concerned not only in walking and standing, but in almost every movement of the body (as the head is always moved at the same time). Some interesting observations (Herzfeld, Kreidl, Wanner, Passow, Bárány, etc.) show that when the semicircular canals on both sides are absent, rotation produces neither giddiness nor loss of balance. The examination of deaf-mutes has had similar results (Bezold, Wanner). In unilateral destruction of the labyrinth, physiological nystagmus is said not to appear during rotation towards the affected side (Wanner). Passow did not obtain definite results of this kind. See the careful discussion of this question by Bárány. Lesions of the vestibular nerve or Deiters' nucleus may have the same effect as lesions of the semicircular canals. Its close connection with the nerves of the ocular muscles explains why paralysis of these nerves may also give rise to vertigo, but in such cases other factors are usually also concerned (see below). The organs innervated by the vagus (stomach, larynx) are also frequently the origin of symptoms of vertigo. In addition to the toxic factors sometimes present, the relations which Edinger and others suppose to exist between the vagus and the cerebellum may also play a part.

It is easy to understand that rapid rotation of the axis of the body, abrupt change of the position of the body, or sudden alteration of our relations to space (swinging, riding on a "merry-

go-round "), etc., will give rise to vertigo. Mach and Breuer have specially helped us to understand this symptom more clearly by pointing to the alterations of tension thus produced in the endolymph, and to the mechanical influence of this upon the auditory cilium. These processes are associated with excessive functioning of the organs which maintain equilibrium. Some objections to this theory have, however, been raised (Eschweiler, Passow).

The interesting symptoms of *galvanic* vertigo, known to Purkinje, and subsequently more fully studied by Hitzig and others, are of practical significance, as in galvanic treatment of the head they may occur during improper use or dosage of the current. The principle is that when the galvanic current is passed transversely through the head, the patient sways towards the side of the anode, when the current is closed and when it is opened, towards the side of the cathode. When the current is still stronger, nystagmus also occurs. According to Babinski (*R. n.*, 1902), when under normal conditions the current is closed the head inclines towards the side of the anode; in unilateral affections of the ear it always leans towards the affected side; in bilateral affections the symptoms of galvanic vertigo can only be elicited with difficulty, etc. Napieralski (*Thèse de Paris*, 1901) has also studied the question, and Mann (*Med. Klinik*, 1907) practically agrees with Babinski's views. He attributes the symptom to a unilateral lesion of the vestibular apparatus. Bárány has shown that syringing the ear with cold or hot water produces irritation of the vestibular apparatus, which gives rise to disturbances of equilibrium and nystagmus, a reaction which is absent when the labyrinth is destroyed.

Hitzig speaks of *systematic* vertigo when the illusory movements of one's own body or of surrounding objects follow a certain direction (rotatory vertigo), and of *non-systematic* vertigo when there is a diffuse feeling of giddiness.

The false projection of the field of vision in paralysis of the ocular muscles is a common *cause* of vertigo. It may therefore accompany diplopia, but may be absent or very transient. The view that vertigo is *always* due to an altered function of the ocular muscles (Mendel [1]) is certainly not correct. This theory is founded on the idea that the oculomotor nucleus, which, according to the investigations of Shimamura [2] and D'Astros, is not at all favourably situated as regards its blood-supply, is most sensitive to every disturbance of the intracranial circulation. Unaccustomed positions of the body, and in particular rapid *revolving* and *swinging* movements, bring on vertigo in most people. Looking down from "giddy" heights has the same effect. Severe vertigo is usually accompanied by nausea and vomiting.

Vertigo is a common symptom in organic brain diseases in which there is *increase of intracranial pressure*, especially therefore in tumour. Tumours of the cerebellum and its peduncles, and indeed all *lesions of the cerebellum* and of the *centres and tracts for co-ordination*, are particularly apt to produce vertigo. Hæmorrhage or softening at any part of the brain may bring on an attack of vertigo, but as a rule the attack is not repeated unless the structures concerned in maintaining equilibrium are affected. *Any sudden alteration in the circulatory conditions in the brain* may have this effect. *Diseases of the vascular system*, accompanied by persistent impairment and retardation of the intracranial circulation, *e.g. atheroma*, are the most common causes of vertigo. In these cases single attacks of vertigo occur, in addition to a persistent dazed, giddy, confused feeling.

Acute anæmia and congestive hyperæmia of the brain both cause vertigo. Women are specially liable to suffer from it at the *climacteric*.

Attacks of giddiness occur in almost every case of *disseminated sclerosis*. The patient suddenly loses his balance, possibly to such an extent that he falls to the ground. A chronic condition of giddiness is less common.

Vertigo sometimes forms an *equivalent* or an aura of an *epileptic attack*.

[1] *B. k. W.*, 1895. [2] *N. C.*, 1894.

In rare cases there may be continuous giddiness between the paroxysms (Hitzig). It may also apparently take the place of an *attack of migraine.*

The most important toxins which produce vertigo are *alcohol*, *nicotine*, and *caffeine.* Uræmic vertigo is due to autointoxication. Gastric disorders are a common cause (vertigo a stomacho laeso). Indigestion or overloading of the stomach may bring it on, and it is a well-established fact that intestinal parasites may cause, and removal of a tapeworm may cure, attacks of vertigo. Constipation is another frequent cause. Stomachic vertigo may take the form of true rotatory giddiness (Trousseau).

Injuries to the head are often followed by giddiness, which may be the only persistent symptom, even when there is no marked lesion of the brain.

The forms of vertigo connected with *diseases of the ear* are of very practical interest, and they may arise from any disease of this organ, or even from an accumulation of wax in the external auditory meatus (Toynbee). But in a special group of cases the aural vertigo is almost constantly associated with acoustic symptoms, in such a typical way that Ménière was able, in 1861, to classify them as a distinct disease.

In these cases the vertigo, at least at first, comes on in paroxysms, and it may be so violent that the patient suddenly *falls to the ground* as if felled by an invisible hand, and lies there *stupefied*, or possibly for a moment unconscious. He then gradually recovers. During the attack he feels as if he were revolving in a circle, as if all the objects round him were moving, or as if the ground were sinking under his feet, etc. Sometimes he falls in a certain direction. *Nausea* and *vomiting* are usually also present, and they may last for some hours. The vomiting may be associated with diarrhœa, and headache is a common symptom (Jackson, Lucæ, Schwabach). More rarely facial paresis develops during the attack (Charcot, Frankl-Hochwart, Oppenheim). The patient looks pale and wasted (refer, however, later to my observations upon the vasomotor form which is accompanied by marked flushing of the face), and the skin is covered with cold perspiration. The aural symptoms, however, are the most characteristic. As a rule there is more or less marked deafness with diminution of bone conduction in one or both ears, and almost always *persistent, incurable tinnitus aurium.* The tinnitus usually becomes louder, just before and during the paroxysm of vertigo, and alters in character, becoming higher and shriller. The deafness may, though it does not always increase. I have found this symptom absent in a few otherwise typical cases. Heermann [1] noted absence of tinnitus in one case, and Frankl-Hochwart refers to this as not being very uncommon. He has found that deafness is not an absolutely constant symptom (vestibular vertigo with an intact cochlea). *Nystagmus* and diplopia sometimes occur. When I have been able to examine the patient during an attack I have always found nystagmus, usually when looking towards the affected ear. Other writers think it appears as a rule towards the unaffected side. The attacks may recur daily or at intervals of weeks and months. At a later period of the disease chronic vertigo is frequently permanent, with transient exacerbations, especially on coughing, turning the head, etc. (status Ménière). This may be so severe as to cause the patient to be confined to bed. Other characteristic signs of a unilateral affection of the labyrinth may be present in addition to the paroxysms.

[1] "Über den Ménièreschen Symptomenkomplex," Halle, 1903.

I have lately seen a case which seemed to prove that there might be a mental equivalent of Ménière's disease in the form of a condition of confusion associated with impairment of the power of speech, but we require further evidence on this point.

In the cases first described by Ménière, the disease had an apoplectiform onset in persons hitherto free from all ear disease. The term Ménière's disease is now applied to the much more common cases in which the symptoms become superadded to an existing ear disease and develop in a more chronic way. Frankl-Hochwart and Politzer therefore prefer the term Ménière's syndrome. Moll, Smith, and others are of the same opinion.

Although this syndrome has been observed in diseases of the middle-ear (Gellé), and even in a few cases of disease or syringing of the external auditory canal, the clinical symptoms as well as the published results of post-mortem examination (Ménière, Politzer, Gruber, Moos, Steinbrügge, etc., and the recent pathological investigations of Pineles-Alt, Schwabach, Sharkey, and Politzer [1]) point to the disease being localised in the labyrinth. In the typical form described by Ménière the cause was apparently, as a rule, hæmorrhage in the labyrinth. This might occur in healthy persons, or be due to syphilis, leucæmia, gout, and other general diseases. Injuries (*e.g.* fracture of the petrous bone, surgical lesion of the semicircular canals) and inflammations may be causes. The latter may be either primary or secondary. Thus labyrinthine inflammation may arise in the middle ear and may be of a specific, possibly also of an epidemic-meningitic origin. Gruber states that exudative processes in the adnexa of the labyrinth (aqueduct, recessus Cotugni) may cause increase of the endolymph, or, in cases where there is displacement of the outlet of the sacculus, impaction of the labyrinth, and may thus give rise to Ménière's syndrome. Frankl-Hochwart has shown that this condition may develop in the course of so-called multiple neuritis of the cranial nerves. It apparently occurs very rarely, if at all, in acute diseases of the ear (Heermann, Frankl-Hochwart).

Lesion of the cochlea is the cause of the deafness, and lesion of the semicircular canals apparently produces the vertigo and loss of balance. The intimate relations between the vestibular nerve and the nuclei of the ocular nerves probably explains the ocular symptoms as stated above.

I have been surprised to find that these and other forms of vertigo very often begin during the night, when the patient turns from one side to the other, especially if he does so abruptly, as one is apt to do in sound sleep.

I have found in a number of cases that Ménière's syndrome appeared in persons suffering from atheroma of the vessels, and cases examined post mortem have shown disease of the vessels leading to the inner ear. Finkelnburg (*M. m. W.*, 1906) has measured the blood-pressure in vertigo. The disease may also follow some injury to the head; I have seen it occur in telephone-operators after a shock from a strong current (see p. 1169).

Symptoms allied to those of Ménière's disease may occur in hysteria and neurasthenia or may represent the aura of an epileptic attack. In a few of my cases the Ménière's symptoms seemed to correspond exactly

[1] Bibliography in Frankl-Hochwart.

to neurasthenia and to be produced by a vasomotor disturbance due to this. I have been specially struck with the fact that *in cases of ear disease, neurasthenia may act as the exciting cause of the development of attacks of vertigo of the nature of Ménière's syndrome.* In one such case, which I have examined along with Jansen, he found only catarrh of the tubes, and agreed with me that the increase of pressure in the labyrinth which gave rise to the attacks of vertigo, had been brought on by the vasomotor disorders caused by neurasthenia. I have occasionally had the opportunity of personally observing such cases and of finding marked vasomotor disturbances. A purplish flush in the face and neck was specially common. In such cases the head was maintained in a certain position ; nystagmus was present, and it seemed to me that there was some limitation of the conjugate movement towards the side of the unaffected ear. I have several times found that in these cases the neuropathic constitution was indicated by a congenital hyperæsthesia of the organs which control the equilibrium, causing giddiness in dancing or looking from a height, etc. Lacharrière,[1] Gescheit,[2] Möller, and others think the origin is an angio-neurotic one. Traumatic neurasthenia may also give rise to this symptom. Lucæ points out that vertigo is a much more constant symptom in traumatic affections of the labyrinth than in other diseases of this organ. Frankl-Hochwart terms the affection pseudo-Ménière, when it occurs in cases with no aural changes. Here the symptoms are probably caused by vasomotor disorders, to which the toxic form of *aural vertigo* is also most likely due. Ménière's syndrome has also been observed in tabes (Charcot, Pierret, Bonnier, Oppenheim). Some persons become giddy when they hear high-pitched tones.

Vertigo is less commonly connected with *nasal diseases.* It may be caused by swelling of the mucous membrane of the nose, of the corpora cavernosa, etc., and may be relieved or cured by treatment of these conditions. *Periodic swelling* of the nasal mucous membrane is apt to occur in neurasthenic persons ; it is due to *vasomotor* disorders and may produce vertigo.

In a few rare cases vertigo has been seen to follow digital examination of the anus (Leube), pressure upon the testicles (Soltmann), or introduction of a bougie into the urinary passages. But as any sudden disorder of the consciousness is apt to be termed vertigo by the laity, it is doubtful whether these were actually attacks of true vertigo.

A *laryngeal vertigo* has also been described, but it is very rare, and hardly answers to the term vertigo. A patient suffering from some laryngeal trouble is suddenly affected with some mental confusion, and *apoplectiform* or *epileptiform* symptoms, which precede a feeling of burning in the larynx and sometimes slight coughing. Loss of consciousness during laughing, mentioned on p. 673, should also be mentioned here.

Vertigo is an exceedingly common symptom of *nervousness*, especially of *hypochondriacal neurasthenia*, and hardly any other symptom can so easily be brought on by *exaggerated self-observation.* I believe that everyone can bring on a feeling of vertigo by concentrating his thoughts upon the process of loss of balance and by calling up the recollections of giddiness, which are familiar to everyone. Neurasthenic individuals have this power in a still greater degree, and in them the fear and thought

[1] *A. f. Ohr.*, xxvii. [2] *B. k. W.*, 1902.

of vertigo are quite sufficient to produce it. In a few cases under my care any attempt at mental work immediately brought on giddiness. It is often the result of over-strained mental activity (Silvagni). In such cases the attacks are usually slight, but neurasthenia may also produce severe attacks on account of the vasomotor disturbances in the labyrinth and the other parts concerned in maintaining equilibrium (as described above) to which it gives rise.

Chronic neurasthenic vertigo is very rare. In two such cases which I have seen, it was peculiarly distressing on account of its intensity and constancy. The patients were never for a moment free from it; in one case (the daughter of consanguineous parents) there was some alleviation during a carriage drive or a railway journey; all the surrounding objects made illusory movements which caused her to make compensatory movements with her body. Even her memory pictures and dream images were constantly in motion. A family form of vertigo also occurs (Oppenheim, Lannois).

An endemic onset of severe vertigo, ptosis, and a kind of paralytic weakness of the muscles of the extremities and neck (sometimes accompanied by diplopia, visual disturbances such as transient amblyopia, photopsia, etc., trouble in swallowing, weakness of the masseters or the muscles of the lower jaw, etc.) has been observed in the canton of Geneva, which has been attributed to miasmatic influences (the air of stables) (Gerlier[1]), and has been named *vertige paralysant*. The paralysis is always flaccid. The affection comes on in paroxysms which last for a few minutes, the sufferers being quite well in the intervals. Gerlier describes three types, according as there is merely ptosis, or ptosis and paralysis of the neck muscles, or in addition, weakness of the extremities and uncertainty of gait. Muscular activity often brings on the attacks. The nature of the condition has not yet been carefully investigated. The prognosis is good. I have been at some trouble to try to see cases of this kind in Geneva, and have been assisted by Ladame, but unfortunately our efforts were not rewarded. Miura[2] has described by the name of *kubisagara* a disease which occurs in Japan and is probably identical with Gerlier's vertigo.

The canoe-vertigo of the Esquimaux (Pontoppidan) may be a topophobia.

Finally, there are some cases of vertigo for which no cause can be discovered.

The *prognosis* depends mainly upon the nature of the primary cause. The outlook in a progressive, incurable brain disease need not be discussed. But it should be noted that the vertigo of arterio-sclerosis may be a temporary symptom and may sometimes entirely disappear. The vertigo of a *diseased stomach* is almost always incurable.

Ménière's disease may resist all treatment, but it is occasionally cured or greatly relieved. Recovery is sometimes simultaneous with the onset of deafness. In two of the most severe cases which I have seen and in which I gave an unfavourable prognosis on account of the existing arterio-sclerosis, recovery has taken place and has now lasted for years. Frankl-Hochwart[3] has recently studied this subject and has concluded from wide experience that the prognosis of Ménière's vertigo is very favour-

[1] *Rev. méd. de la Suisse romande*, 1887-88. [2] *Mitt. d. med. Fakult.*, Tokio, 1896.
[3] *Jahrb. f. P.*, 1905

able, as recovery has taken place in forty out of seventy-four cases. The prognosis of nasal vertigo is very hopeful.

Treatment should in the first place be directed to the cause. If the vertigo is due to hyperæmia of the brain, it may be cured by *aperients*, *hot foot-baths*, or by single or repeated *blood-letting*. Treatment of a gastric disorder, the avoidance of errors in diet, the regulation of the bowels by treatment at Carlsbad, Homburg, Marienbad, Kissingen, or by the use of enemata, etc., cure many cases of vertigo. *Tape-worms* should be removed in the ordinary way. If arterio-sclerosis is the cause, *iodide of potassium* or *iodide of sodium*, *iodipin*, etc., have sometimes a good effect. A combination of these drugs with ergotin or digitalis—digitalis ½ gr. pot. iod. 15 grs. (Hitzig)—or with diuretin has been recommended. It may be combined with the bromides if a symptomatic effect is desired.

In Ménière's disease the aural disease should be treated. In addition to direct or surgical treatment, the advisability of the air douche, vibratory massage, etc., may be considered, but Heermann is right in warning us against injudicious attempts to treat this affection by various aural methods. Excision of the semicircular canals has lately been tried as a remedy for aural vertigo, and has been described as successful in the cases of Lake,[1] Milligan,[2] and Isemer.[3] The vertigo may sometimes be relieved by the use of the bromides or belladonna. According to Charcot's accounts, much benefit may follow systematic administration of quinine, which Ménière himself prescribed. It must be given over a long period in daily doses of 9 to 15 grs., at first for two to three weeks, and then again after an interval. The condition is at first intensified (according to Charcot the quinine is fighting the disease), and then it gradually improves. Gilles de la Tourette gives still larger doses. I have occasionally seen good results from it, and even recovery in a few severe cases, but aurists are opposed to its use from the fear that it may aggravate the deafness. Frankl-Hochwart also disapproves of quinine. Charcot would accept the deafness, on account of the disappearance of the vertigo which accompanies its onset, but this is not a very happy prospect for the patient. I have in a few instances seen very good results from blood-letting which Lucæ [4] has recommended, especially in cases in which the attack was accompanied with symptoms of congestion. Salicylate of soda has also been used. Hirt recommends *injections of pilocarpin* (5 to 8 drops of a 2 per cent. solution every second day, for a week), and others have found it good. Gruber prescribes tinct. of arnica and tinct. of nux vomica; Gowers gives a combination of bromide with gelsemium and extr. hyoscyami, and Bürkner [5] bromide of potassium with iodide of potassium. Babinski [6] has found lumbar puncture successful in many cases. He withdraws a few cubic centimetres (from three or four to twenty) at one or more operations. The improvement in the vertigo follows within a few days, and the noises in the ears are often relieved, etc. According to his results and Schönborn's [7] communication, this measure, which should be used with every precaution, deserves to be recommended. Ebstein has found the use of oil enemata to be of great service. Sternberg [8] reports having arrested the attacks by enemata of

[1] *Lancet*, 1904. [2] *Brit. Med. Journ.*, 1904. [3] *M. m. W.*, 1907.
[4] Eulenburg's "Realenzyklop.," third edition. [5] *D. w. W.*, 1905.
[6] "Annal. des mal. de l'oreille," 1904; see also Lumineau, *Thèse de Paris*, 1903.
[7] *Med. Klinik*, 1906. [8] *D. m. W.*, 1906.

sod. bromid. 0·6 (say 10 grs.), antipyrin 0·6 (10 grs.), opii. gutt. 20, muc. gumm. acac. 60·0 (say oz. fl. 2). The gymnastic exercises (circular movements of the head, etc.) recommended by Urbantschitsch [1] can only be useful in psychogenic vertigo of mental origin. Donath states that he has had excellent results from the use of the constant current, and Lacharrière, Gescheit, and Bloch [2] have found it good in the angioneurotic form. Parry improved the condition by the use of a seton. A stay in the mountains is recommended by Frankl-Hochwart, and I have found residence in the hills at a moderate height very successful in a few cases.

Treatment of hypertrophy of the nasal mucous membrane may have a curative effect.

If the symptoms are of neurasthenic origin, the treatment should be practically similar to that of neurasthenia. The symptoms may be treated by the use of the bromides, phenacetin, hydrobromate of quinine, nitroglycerine, galvanisation of the brain, faradisation of the neck or the soles of the feet. Static electricity may be applied in a similar way to distant parts of the body. Hydrotherapy is often found to be an excellent remedy. I have sometimes found a hot foot-douche to have a good effect in very persistent cases. In a few cases it may ultimately be advisable to use a seton. The vertigo sometimes resists all treatment, especially when it takes the very rare form of chronic neurasthenic vertigo.

Glossodynia (Neurosis of the Tongue)

This symptom—it is doubtful whether we can call it an independent disease—consists in paræsthesiæ, generally a burning, prickling sensation, confined to the tongue or extending to the mucous membrane of the cheeks, jaws, and lips. It may occur in paroxysms or be constantly present, and it may disturb the sleep. It is not on the whole a very common symptom, and generally affects women of advanced age. The teeth are almost always absent. The sufferer is usually of a neuropathic constitution. A gouty diathesis is said to create a predisposition. In several cases *cancrophobia, i.e.* fear of cancer, was present, but it was impossible to decide whether the hypochondriacal fear was the cause or the effect of the paræsthesiæ. There were no objective symptoms in my cases, except occasionally a transient or persistent diminution of the sense of taste. The condition is chronic and may last for years. So-called *pruritus senilis linguæ* also belongs to this type. Paræsthesiæ and pain in the tongue may appear in the initial stage of tabes and paralytic dementia.

Similar disturbances may occur elsewhere. Thus I have found persistent urethrodynia in one of my cases.

Glossodynia should not be confused with xerostomia (dry mouth), a condition of abnormal dryness of the tongue, mouth, and pharynx of nervous origin.

Glossodynia should be chiefly treated by psychotherapy. Local painting with cocaine, nitrate of silver, etc., has been recommended, and electricity may also be tried.

[1] *W. kl. W.*, 1901.

[2] *Prag. med. Woch.*, 1903.

Epilepsy, Falling Sickness (Morbus Sacer)

Literature: Nothnagel, in Ziemssen's "Handbuch," xii.; Gowers, "Epilepsy and other Chronic Convulsive Diseases," etc., London, 1881; Voisin, "L'Épilepsie," Paris, 1896; Féré, "Les Épilepsies et les Épileptiques," Paris, 1890; Binswanger, "Eulenb. Realenzyklop.," third edition, article on Epilepsy; Binswanger, "Die Epilepsie," Nothnagel's "Handbuch," xii., 1, Wien, 1899 (containing good review); Pini, "L'Epilessia," Milano, 1902; Turner, "Epilepsy, A Study of the Idiopathic Disease," London, 1907.

Amongst the compilations of recent years see those of Biro, *Z. f. N.*, xxiii.; Fleury, "Rech. clinique sur l'Épilepsie," Paris, 1900; Sarbo, "Der heutige Stand," etc., Berlin, 1906; Finckh, *A. f. P.*, xxxix.

Epilepsy is a disease which in its fully developed form is characterised by *attacks of unconsciousness associated with convulsions*.

According to the unanimous experience of writers on the subject, such as Herpin, Moreau de Tours, Berger, Neckas, Echeverria, Gowers, Lange, *heredity* is the most important, or one of the most important of the many *causes of the disease*. A number of conditions are capable of producing it, but apparently they make their influence felt chiefly when the morbid predisposition is already present. A hereditary tendency to disease may be ascertained in about one-third to one-half of the cases, in 35 to 40 per cent. according to Binswanger, and in nearly three-fourths according to Finckh. In about two-thirds of these cases the heredity is direct.

Epilepsy produced experimentally in animals may also be transmitted (Brown-Séquard, Luciani), although objections have been raised to this opinion (M. Sommer, etc.).

A *toxicopathic* condition is another predisposing factor. Chronic alcoholism and chronic lead-poisoning in the parents may lead to epilepsy in the children.

Poisons may directly bring on epilepsy. *Alcoholics* (brandy and absinthe drinkers) are frequently epileptic.

The importance of this factor is very differently estimated. According to the experience gained in the Charité (Moeli, Thomsen, etc.), alcohol is one of the principal causes. Other writers, *e.g.* Wildermuth,[1] could only trace a small proportion of the cases to chronic alcoholism. Wartmann (*A. f. P.*, xxix.) found 206 drunkards among 452 male epileptics, but half of these had been epileptic from their youth, and had only become alcoholics at a later stage, and in the other cases the excessive use of alcohol was associated with other injurious circumstances which undoubtedly played some part in the causation. In many cases hereditary mental degeneration is the cause both of the alcoholism and of the epilepsy. The reader should refer to the papers by Neumann ("Dissert.," Strassburg, 1897) and Bratz and Siebold (*Psych. neur. Woch.*, 1906) on this subject.

It is said that drunkenness during the time of conception may lay the foundation for epilepsy in the child (Esquirol). Attacks which can hardly be distinguished from epileptic fits may result from chronic lead-poisoning. There is less certainty as regards the epileptic nature of the attacks which occur in poisoning from other drugs (cocaine, antipyrin, arsenic, chloroform, ether, physostigmin, etc.). Marburg observed an interesting case in which epilepsy developed after many years' use of roasted coffee-beans (25 to 180 grs. per day), but the case is as singular as another in which chronic nicotine poisoning was thought to be the cause. Rosenthal saw convulsions of an epileptic type as a symptom of camphor poisoning, and Salomonson saw them follow the use of 0·5 to 1·0 g. (say 7·5 to 15½ grs.)

[1] "Die Ätiol. d. Epilepsie," Festschrift, Stuttgart, 1897. See also *N. C.*, 1897.

of camphora monobromata. Schlesinger[1] describes convulsive conditions after the use of theophyllin. Epileptic convulsions have occasionally been observed in recent years to follow lumbar anæsthesia with *stovain*. Uræmic, diabetic,[2] and acetonæmic convulsions should be regarded as quite different from epilepsy. It has not been proved for certain that gout can produce true epilepsy. Redlich mentions epilepsy due to vaccination.

There is no doubt as to its relation to *acute infective diseases*, especially scarlatina, and to typhoid, measles, smallpox, influenza, etc. Malaria has also been regarded as a cause (Du Montyel). These diseases are specially apt to lead to epilepsy in children. Numerous cases show that some organic brain disease in childhood, identical with or very similar to infantile cerebral paralysis, may, without producing typical paralytic symptoms, lead to the development of epilepsy. Some writers, such as Marie and Freud, even go so far as to attribute epilepsy in general to some such organic disease of the cerebral cortex. Redlich[3] also thinks this a very important mode of origin, but there is certainly no justification for this view. Allen Starr[4] is of opinion that epilepsy is entirely due to organic changes.

Among the chronic infective diseases, *syphilis* is a very important cause. In the great majority of cases, it is true, syphilitic epilepsy belongs not to the true, but to a *symptomatic* form, and is merely one of the signs of cerebral syphilis. I think, however, with Fournier, Lancereaux, Trousseau, etc., that many cases show beyond a doubt that ordinary epilepsy may be a consequence of acquired and inherited syphilis. Binswanger and Luzenberger in particular have recently emphasised the importance of the latter, whilst Bratz and Lüth[5] have found it present only in 5 per cent. of their cases.

Epilepsy very often follows some *injury to the head* (a blow or fall upon it). Partial epilepsy is the form generally caused by cortical lesions, and it will not be discussed here. Injuries to the head may, however, give rise to the onset of *true epilepsy* (especially in individuals of hereditary predisposition and alcoholics). Trephining is said to have had this result in a few cases (Robert). There is a form of traumatic cortical epilepsy in which no organic changes could be discovered in the corresponding area of the cortex (Raymond).

Breitung (*D. m. W.*, 1898) reports a singular case in which epileptic attacks came on after prolonged use of douches to the head.

It is certain that the disease may have a *reflex* origin from some peripheral irritation. This connection is most obvious in cases of traumatic *reflex epilepsy*. Many cases show that injury of a peripheral, in particular of a cutaneous nerve, or its irritation by a cicatrix or a foreign body may produce spasmodic attacks of an epileptic nature.[6] Naturally a cicatrix of the scalp and possibly a lesion of the meninges may give rise to reflex epilepsy. Many facts seem to indicate that a stimulus arising from any part of the body may have this effect. Observations upon this point have

[1] *M. m. W.*, 1905. [2] See on this subject Soetbeer, *M. f. P.*, xxii., Erg.

[3] *A. f. P.*, Bd. xli.; *W. kl. R.*, 1905; see also "Epilepsie und Linkshändigkeit," *A. f. P.*, Bd. xliv.

[4] *Journ. Nerv. and Ment. Dis.*, 1904. [5] *N. C.*, 1900.

[6] Of the papers treating specially of this form, we would refer to those by Seeligmüller (Festschrift, Leipzig, 1897) and Urbantschitsch (*W. kl. W.*, 1906).

not led to undisputed results, but epilepsy has been attributed to diseases of the *nose* and its *accessory cavities*, of the *stomach*, *liver* and *uterus*, to *intestinal worms*, *laryngeal polypi*, *foreign bodies* in the *ear*, *phimosis*, and even to *errors of refraction* and *carious teeth*. Although these are still somewhat hypothetical opinions, we should always bear in mind the possibility of such a relationship, which is sometimes clearly demonstrated by the results of treatment.

Frey and Fuchs [1] have made a careful clinical and experimental study of this subject and have concluded that, given the existing predisposition, such irritations are quite capable of exciting the attacks. The predisposition may be congenital or due to the age of the child, or it may be acquired from some injury to the brain resulting from focal disease, trauma, etc. This is the point of view which I have already advocated.

It is very doubtful whether the attacks caused by intestinal parasites are really epileptic, but Peiper's theory that such parasites produce toxins in the organism has brought the existence of an epilepsy of this origin more within our comprehension.

We do not yet know whether the convulsions which occur during *dentition* should be regarded as reflex epilepsy.

It has been stated (Bastin) that epilepsy may be the result of affections of the thyroid gland. Consult this author also as regards its relation to tetany.

Masturbation has been said to be a cause of epilepsy. I have seen a few cases which would indicate this relationship, although early masturbation is, of course, usually a sign of the neuropathic constitution.

The statement that epilepsy may be brought on by coitus interruptus should not be taken seriously.

Attacks which are possibly of an epileptic nature sometimes occur in the course of *heart disease* (Lemoine, Rosin,[2] etc.). They have several times been observed in aortic stenosis and stenosis of the cardiac orifices; but some writers, such as Leser,[3] have recently disputed this connection. The epilepsy which develops in advanced age (epilepsia tarda, senilis) has also been attributed to atheromatous disease of the heart and vessels, but there is no unanimity of opinion among the writers who have studied this question (Redlich,[4] Mendel,[5] Mahnert,[6] Lüth, Stintzing, Binswanger, Bratz, Parisot, Schupfer, Masoin, Medea,[7] etc.). In such cases *disturbances of the circulation* are obviously the immediate cause of the attack. Bresler [8] assumes that epilepsy may also be produced by chronic affection of the kidneys. Smith attributes a special form of alcoholic epilepsy to the heart. Stokes had already stated that epileptiform attacks may occur in bradycardia (see p. 675), and I have occasionally observed them in *aortic aneurism*.

Moreover, *mental excitement* may give rise to epilepsy. *Fright* is specially apt to have this effect (Gowers), although it more frequently brings on an attack of hysteria, but there is no doubt that it also plays a part in the causation of epilepsy. It acts most powerfully upon predisposed individuals, but it is probable that violent shocks of this kind may undermine the normal brain. The remarkable increase of the acustico-

[1] *Obersteiner*, xiii., 1907.
[2] *W. m. Pr.*, 1893.
[3] *Arch. bohém.*, 1904.
[4] *W. m. W.*, 1900.
[5] *D. m. W.*, 1893.
[6] *W. m. W.*, 1897.
[7] *Boll. Poliamb. di Milano*, 1902.
[8] *Psych. Woch.*, 1899.

motor excitability in spastic diplegia (*q.v.*) is very instructive in this respect. Anger may excite the convulsions, and coitus has been said to do so. Emotional excitement in the mother during pregnancy has been regarded as a cause (Voisin, Féré).

No age is exempt from this disease, but in most cases, indeed in three-fourths of the whole, it commences before the age of twenty. The time of puberty is apt to create a predisposition, and about half the cases occur between the ages of ten and twenty. Early childhood is also a susceptible period, whilst the disease seldom occurs after the age of twenty and grows rarer with advancing age.

Symptomatology.—Epileptic fits are the chief and often the only element of the disease. During the intervals between them the patient is often absolutely well; but in some cases, and especially in the later stages, his health may be constantly affected.

There are three different types of epileptic attacks: (1) the severe attack (major epilepsy or grand mal) or *epilepsia major seu gravior*; (2) the mild forms, viz., *petit mal* or *minor epilepsy*; (3) the *equivalents* of epileptic attacks.

The major attack may come on suddenly. In many cases there are premonitory symptoms, though seldom definite disturbances of the general health. These prodromata are vertigo, fulness in the head, depression, tremor, excitability, which may last for *hours* or even days before the attack comes on. There may also be muscular twitchings in one extremity or more widely diffused (Reynolds, Charcot).

These twitchings may exist for a long time in children before the onset of the true epileptic attacks (Ballet, Dide, etc.), or they may be the only symptom of the epileptic diathesis (Ballet-Bloch, *R. n.*, 1903). Pruritus, urticaria, erythema, spasms of sneezing, tinnitus aurium, photophobia, etc., are less common prodromata (Féré).

More often there are symptoms which occur some *seconds* or *minutes* before the attacks or at their onset. These symptoms, known as *aura*, are of many kinds, but the same aura is almost always repeated before each attack; the patient is practically always warned of the approaching attack by the same symptom. The aura may, however, only usher in one part of the attack, while the other parts commence immediately with loss of consciousness.

The characteristic symptoms of the aura may be of *motor*, *sensory*, *secretory*, *vasomotor*, *visceral*, or *psychic* nature.

The *motor* aura consists in twitchings which usually affect a single group of muscles in one limb, and may then spread in the same regular way as in cortical epilepsy. But as a rule the loss of consciousness and the general convulsions follow so quickly that the aura remains limited to a few muscles, or to one extremity. Bilateral symptoms of motor irritation may also precede the onset of the attack. Complicated movements, such as running forwards, backwards, or in a circle, scratching gestures, etc., are less common.

The aura may also consist of speech disorders of an aphasic (Ross) and dysarthric nature (Clark [1]).

A *sensory* aura more frequently occurs. There may be paræsthesiæ, *e.g.* a feeling of formication or numbness in one extremity, and these may spread in an upward and downward direction before the patient becomes

[1] *Rev. de Méd.*, 1883.

unconscious. Or there may be a peculiar sensation ascending from the stomach or abdominal region, or from the whole lower part of the body. Globus may also form an aura. There is sometimes a curious feeling which the patient cannot describe, or *pain*, often in the epigastric region or passing upwards from the abdomen, or in rare cases dull pain in the head.

Some sensory perception, *e.g.* a peculiar *smell* or *taste*, or some sensation in the *ear* and very often in the *face*, frequently represents the aura. The *acoustic* aura consists in the hearing of a sound, a noise (whistling, buzzing), or less often a word or a melody, or in loss of sight. The *visual* aura is usually represented by the sight of a colour or spark, sometimes of complicated pictures, *e.g.* the figure of an animal, a man, a caricature, a beautiful landscape, etc., or by loss of sight. In other cases the patient has the feeling that objects around him become larger or smaller, approach or recede, or rotate in a certain direction. These sensations are usually accompanied by a feeling of vertigo.

The *vasomotor* aura is characterised by sudden pallor, which may be limited to certain sites, by flushing of the face or one side of the body, or by red spots appearing on certain parts. The aura in rare cases takes the form of *respiratory disturbances*, such as singultus, inspiratory spasm, fits of yawning, etc., or it may occasionally be of a *secretory* nature, consisting usually in *profuse perspiration*.

The *mental* symptoms are: anxiety, timidity, depression, the remembrance of some past experience or situation, or the patient may have a kind of *memory hallucination*, viz., a feeling that he has already lived through his present experiences. The psychic aura may develop into *pre-epileptic insanity*, which may simulate delirium (see below).

The Attack.—After an aura, or more frequently without any premonition, the patient suddenly falls down *unconscious*. At the same time there is *tonic, tetanic contraction of the muscles*, including those of respiration, so that the breathing stops. The sudden forcible expiration of the air along with the simultaneous closing of the glottis may give rise to a short, deep, or shrill cry, which is in many cases the first signal of the attack. There is sometimes a gurgling, inspiratory noise.

The face, at first pale, soon becomes red, then purple, and has a bloated, distorted look; the eyes are shut or open, and the eyeballs seem to be starting out of their sockets; the pupils are dilated and the *pupil reflex is absolutely gone*. The head is drawn backwards or to one side (in rare cases the whole body is twisted), the eyeballs are usually rotated towards the same side or turned upwards, the arms spasmodically flexed or extended, the hands clenched or held in the writing position, whilst the thumbs are adducted and usually pressed in. The legs are as a rule stiffly extended, but they may be flexed. In rare cases the head, trunk, and extremities are brought into a position of flexion, producing a crouching attitude. The tongue is now or later caught between the teeth. Foam, saliva, and mucus dribble from the mouth. Tonic contraction of the muscles may lead to discharge of urine and fæces. The stage of *tonic spasm* lasts from some seconds to half a minute. During or at the close of the spasm the tonic muscular contractions may be combined with tremor. Then follows the period of *clonic contractions*: at first, there are short twitchings followed by strong contractions of the muscles of the head, trunk, and extremities. These increase in intensity and in rapidity of

succession, and may be so violent as to cause injury of the soft parts, bones, and joints.

Dislocation of the humerus is a not uncommon occurrence. I have seen a case in which each attack caused bilateral dislocation of the humerus. Dislocation of the lower jaw has also been observed (Stanley). A complete summary of the injuries occurring during epileptic attacks will be found in a paper by H. Fischer (*Arch. f. P.*, Bd. xxxvi.).

When heart disease is present, an embolism of the cerebral arteries may occur during the attack (Broadbent, *Br.*, 1903).

The clonic spasms of the jaw muscles often cause *biting of the tongue*. The eyeballs also become involved in the twitching movements. The onset of the clonic contractions is accompanied by the entrance of air into the lungs; the cyanosis passes off and the breathing becomes noisy and rapid. The pulse is generally abnormally rapid. The whole body may be covered with perspiration. At this stage there is often discharge of urine, less often of fæces or seminal fluid. Götze has observed fæcal vomiting in one case.

This stage lasts from half a minute to five minutes. Towards the end the convulsion becomes less rapid and is confined to a smaller number of muscles. The attack seldom comes to an end with the cessation of the convulsions. There is usually a third stage, during which the *coma* persists; the patient may open his eyes for an instant, but he is still dazed, and the coma often passes into *sleep*, which may last for some hours. This sleep cannot be distinguished from the sleep of health; the patient may be wakened out of it, but he is then dazed and complains of headache. When he is roused out of his sleep, a kind of "crepuscular condition" may develop, in which he moves and acts automatically (Hermann [1]). The breathing remains stertorous during this stage. Vomiting may occur. Some epileptics vomit after each attack.

In these typical cases there is complete amnesia for the time of the attack; in less common cases it includes a period preceding the attack (Féré, Séglas, Maxwell [2]). Alzheimer [3] mentions amnesia of longer duration.

The following *accessory symptoms* of the epileptic attack should also be mentioned:—

The temperature is raised as a rule, but only about 0·1 to 0·5° C. (Bourneville). Marked pyrexia only occurs in *status epilepticus*.

The *reflexes* (including the conjunctival and corneal, and in particular the pupillary light reflex) are absent during the attack, and if the tendon reflexes can be tested, they are often found to be absent, but they may be normal or exaggerated (Sternberg, Guichaux [4]). This loss of the reflex excitability may last a short time longer than the attack. I have only once succeeded in finding Westphal's sign to be present before the attack, and after the patient had complained of premonitory symptoms. Babinski [5] found his toe-reflex present during and a short time after the attack. I have occasionally been able, in the few cases which I could examine, to elicit the Oppenheim sign. The "feed-reflex" which I have found in epileptic coma should be regarded as of great importance. Fürnrohr [6] has also found it in some cases. Small ruptures of vessels in the skin and mucous membrane, or circumscribed *hæmorrhages* in the skin and

[1] *Russ. med. Rundschau*, 1902-1903.
[2] "L'Amnésie et les troubles de la conscience dans l'épilepsie," Paris, 1903.
[3] *Z. f. P.*, Bd. liii. [4] *Thèse de Bordeaux*, 1902. [5] *R. n.*, 1899. [6] *Z. f. N.*, xxvii.

conjunctivæ sometimes occur. This well-known symptom has been again recently described by Bourneville [1] and Pfister,[2] and in rare cases it may become very marked and extensive (Pichler, Bychowski [3]). The urine passed during or shortly after the attack, sometimes contains a trace of *albumen*. Lannois found postparoxysmal albuminuria in many cases, Rabow, Fürstner, Binswanger, and Inoyue-Saiki [4] only in a minority of theirs. Polyuria may follow the attack. The amount of urea is sometimes increased. This symptom, which although repeatedly observed has also been disputed, was regularly and undoubtedly present in one of my cases.[5]

The discs, which are pale at the commencement of the attack, become hyperæmic (D'Abundo). Knies found the retinal arteries contracted, and also saw convulsions of the ciliary muscles. L. S. Meyer noted a venous pulse on the retina.

The post-epileptic sleep may be replaced by a condition of difficulty in recollecting things and of confusion, which may develop into pronounced mental disturbance (see below).

In rare cases the attack is followed by motor weakness or paralysis (monoparesis, hemiparesis), or by a speech disorder in the form of aphasia or stuttering.[6] These transient paralyses have been regarded as an expression of exhaustion of the motor cortical centres (Bravais, Todd, Jackson, Clark [7]). There is very occasionally transitory paraplegia (Le Grand du Saulle, Heveroch [8]), possibly associated with hypotonia and loss of the tendon reflexes. Transitory amaurosis and amnesic aphasia have been observed by Schmidt [9] after the attack. In an epileptic of eleven years of age, whom I saw in Bergmann's clinic, a period with a rapid succession of fits terminated in dysarthria and inco-ordination of all the movements, which gradually passed away. I have seen other cases, particularly in childhood and youth, in which the postparoxysmal symptoms of paralysis were very marked after an accumulation of attacks. The more severe and persistent these symptoms, the more probable is it that the epilepsy is not genuine, but symptomatic. This is not the case as regards *concentric narrowing of the field of vision* and *disturbances of the general sensibility*, which, as Thomsen [10] and I [11] have found, frequently follow an attack of genuine epilepsy and persist for a considerable time after it. Féré, Richter, and D'Abundo have found this also. Muskens [12] thinks he found sensory disturbances of segmentary distribution before and after epileptic attacks. *Erythematous flushing* of one or more spots on the skin may be a symptom of a previous attack. Tremor, conjugate deviation, paralysis of the ocular muscles, blindness, deafness, vomiting, diarrhœa and other gastric disturbances, polyuria, salivation, œdema, cutaneous emphysema (Ransohoff [13]), etc., have been described as post-epileptic symptoms.

The convulsion does not necessarily follow the aura. In the cases in which the aura begins in one extremity—notably the hand—and consists

[1] *Progrès méd.*, 1902. [2] *Ärztl. Sachverst.*, 1903. [3] *Medycyna*, 1903.
[4] *Z. f. phys. Chemie*, Bd. xxxvii. [5] *Z. f. kl. M.*, vi.
[6] For an account of the various speech disturbances occurring in the course of epilepsy see Raecke, Bernstein (*M. f. P.*, xvi.), Heilbronner (*C. f. N.*, 1905), Pick (*Ann. méd. psych.*, 1903), and Neisser (*C. f. N.*, 1905).
[7] *Rev. de Méd.*, 1883. [8] *R. n.*, 1902. [9] *C. f. N.*, 1906.
[10] *N. C.*, 1884. [11] *C. f. d. med. Wiss.*, 1884; *A. f. P.*, xv.
[12] *A. f. P.*, xxxvi. [13] *N. C.*, 1904.

in local paræsthesiæ or twitching, it is sometimes possible to prevent the attack by immediately applying a ligature to the extremity above the site of the aura. Rubbing or traction on the muscles has less often this effect. One of my patients, whose convulsion commenced in the hand, could, by suddenly and forcibly over-extending it, suppress the attack.

Some of my patients (male epileptics) report the remarkable fact that they have sometimes been able to arrest the attack by a *strong effort of will*, and by putting forth all their strength to retain their consciousness. These statements, which were published in the first edition of this textbook (1894), have received surprisingly little attention, but have been confirmed by the observations of Roskam, Pick, Campbell, Thomson,[1] and others.

Under the name of *epilepsia procursiva* or *running-epilepsy* (Bourneville, Ladame) a variety of the epileptic attack has been described, in which the patient—as if driven by some blind impulse, of which he is quite unconscious—runs some distance forwards or less often backwards before the actual attack commences, or even without any further development. This form is said to occur in congenitally degenerate and perverted individuals. It may alternate with typical attacks, and there is no justification for assigning it to a special cause. Lannois mentions a "rétropulsive" epilepsy. We should also here refer to the attacks in which the epileptic, seized by some uncontrollable spirit of restlessness, wanders about for hours and days, makes long journeys, behaving apparently in an orderly way, but being in a state in which his consciousness is greatly altered, so that he has afterwards either no recollection, or but a very fragmentary one, of his purposeless proceedings (Lasègue, Le Grand du Saulle, Charcot, Burgl, etc.). Donath does not regard this amnesia as a necessary consequence of the epileptic wandering mania (poriomania). It has been already mentioned on p. 1077, that this symptom may be the result of hysteria and mental degeneration. See also Raecke, *A. f. P.*, Bd. xliii.

In very rare cases the convulsions not only begin in one side of the body, but remain entirely limited to it. The attack is then exactly similar to a cortico-epileptic fit, and this form of true epilepsy can only be distinguished from the *Jacksonian* form by the course of the disease and the accessory symptoms. Status epilepticus may also show this distribution (see below). This Jacksonian type of true epilepsy has been observed as a hereditary or directly transmitted disease (Leenhardt-Novero [2]).

Amongst the incompletely developed forms we should include those in which the tonic or clonic stages are absent, the whole attack consisting of a sudden loss of consciousness (the apoplectic form of Trousseau). These form the transition to:

Minor Epilepsy (petit mal).—Loss of consciousness is the chief and often the only symptom of these attacks. This comes on suddenly or less commonly follows an aura. The unconsciousness usually comes and goes so rapidly, passing off in a few seconds or half a minute, that the patient as a rule does not fall, and resumes his interrupted conversation or occupation as if nothing had happened in the meantime. In many cases he is quite unaware of his attacks. If he is observed during the state of unconsciousness, certain disturbances may be recognised. The face is usually pale, or in rare cases flushed; the eyes are staring; and there are often *slight clonic twitchings*, especially in the lips or tongue (tasting movements [3]), and smacking noises, blinking of the eyelids, or slight twitch-

[1] *Lancet*, 1903. [2] *R. n.*, 1905.

[3] This possibly represents a rudimentary form of the "feed-reflex," which I have described, and is brought on during the sudden loss of consciousness by the saliva going down the wrong way, etc.

ing movements in the extremities. The tongue is not generally bitten, nor is the urine passed involuntarily.

A very transient *condition of vertigo* may constitute the attack, instead of the loss of consciousness, but this so-called epileptic vertigo can hardly be diagnosed unless it is accompanied by other signs (momentary loss of consciousness, loss of urine, rapid twitchings) or by secondary symptoms.

In rare cases the attack may be represented by transient loss of power of speech or comprehension of what is said. I have observed this in several cases, and it has been described by Heveroch (*N. C.*, 1904).

Another variety of the epileptic attack has been reported, which consists of very transient paralysis, sudden giving way of the legs (the knees bending), or symptoms of motor excitement, *consciousness being retained* (Krause, Binswanger, Diehl,[1] Marchand-Olivier [2]). Such attacks can only be rightly interpreted when they occur in addition to typical attacks.

The loss of consciousness sometimes follows, or is replaced by a *condition of dreamlike confusion*, in which the patient moves about, runs away, undresses himself, exposes his genital organs, performs complicated and apparently premeditated actions of which he is quite unconscious, and which he afterwards entirely forgets. Some of the conditions described as *automatisme ambulatoire*, fugues, poriomania, etc. (see p. 1077), belong to this class. It also includes all the remarkable disturbances of the mental life which form an *equivalent of the epileptic attack*, *i.e.* which appear in place of the convulsions and reveal their innate relations by alternating with them and showing a number of characteristic peculiarities.

These *psychic equivalents* cannot, however, be sharply distinguished from the forms of *postepileptic mental disorders* which develop after an attack or a succession of attacks. The same mental disorder which, if it appeared independently, would have the significance of an "equivalent," may follow upon an epileptic attack. Indeed some writers, such as Le Grand du Saulle, and Magnan, refuse to admit the conception of the equivalent.

The various forms of epileptic insanity, specially studied by Morel, Falret,[3] and Sammt,[4] and recently by Raecke,[5] Aschaffenburg,[6] Siemerling,[7] Heilbronner,[8] H. Liepmann,[9] and others, have a number of common characteristics. There is usually a condition of absolute *confusion*, in which the patient speaks disconnectedly and acts unreasonably and without any self-control, running away, setting out on a journey, passing his urine in a room, undressing himself, stealing, etc. After the attack, which generally lasts from a few minutes to an hour, seldom longer, the patient has as a rule absolutely no memory of what has happened.

The amnesia may also be incomplete (Maxwell). Some writers (Gräter, Thomson, Riklin, in *Journ. f. P.*, i. and ii.) have reported that they have occasionally evoked the epileptic amnesia during hypnosis.

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[1] *M. m. W.*, 1901
[2] Abs. in *R. n.*, 1907.
[3] *Arch. gèn. de Méd.*, 1860-61.
[4] *A. f. P.*, v. and vi.
[5] "Die transit. Bewusstseinsstörungen der Epileptiker," Halle, 1903; and *M. m. W.*, 1904.
[6] *A. f. P.*, Bd. xxxvii., and "Die Stimmungsschwankungen der Epileptiker," Halle, 1906.
[7] *B. k. W.*, 1895, and *A. f. P.*, Bd. xlii.
[8] *M. f. P.*, xiii.
[9] "Die deutsche Klinik," etc., 1905.

Epileptic delirium is characterised by paroxysms of *violent excitement of a maniacal nature*, during which the patient is compelled by some irresistible power, under the influence of sensory hallucinations and delusions, to commit violent acts, or he may be seized by an insane desire for destruction and may commit very serious and terrible crimes. These attacks last for hours or days ; they are seldom protracted, but may persist for some weeks or longer (Mörchen [1]). As a rule there is no recollection whatever of the condition, but in rare cases some vague idea may persist in the patient's mind. These mental equivalents are usually sudden in their onset ; but sometimes the patient has premonitory sensations. It is to a certain extent characteristic that the various attacks almost entirely resemble each other. It has been rightly noted that there is never complete loss of consciousness in these conditions, but rather a peculiar kind of disorder of consciousness, consisting of a dreamlike, altered consciousness in which apparently orderly, indifferent, ordinary acts are accompanied by others of a strange, unreasonable, violent nature (Siemerling). The attack may greatly resemble mania, being associated with flight of ideas, etc. (Heilbronner). A condition of great anxiety with terrifying visions may form an equivalent of the epileptic attack (the *petit mal intellectual* of Falret). See also Gowers on psycho-epilepsy [2]).

Post-epileptic insanity is, as we have already said, identical in many respects with the psychic equivalent. It appears in the form of *epileptic delirium*, *stupor* with dreamy confusion, a *condition of dread with horrible hallucinations*, a crepuscular state with persistence of some one idea, etc. Conditions of simple apathy and dementia have also been observed. Post-epileptic insanity may last from a few hours to some weeks.

Another equivalent of the epileptic attack is sudden and causeless *outbreaks of perspiration*, consciousness being normal or impaired. This is a very rare occurrence. A patient suffering from epilepsy may suddenly fall asleep (narcolepsy), and this should also be regarded as an equivalent of an attack (Westphal, Gélineau, Féré, Furet, see p. 1076). The patient cannot be wakened out of this sleep, which may be followed by violent delirium.

In a few cases the epileptic attack is revealed by automatic repetition of a few senseless words, verbigeration, etc. (Cheadle). This condition has been termed *muttering epilepsy* (*épilepsie marmottante*).

A general tremor, associated with an alteration of consciousness, may also represent the attack. Certain forms of angina pectoris, paroxysmal tachycardia, spasm of the glottis, asthma, neuralgia, transient hemiplegia, aphasia (Strohmayer), profuse salivation, gastric disorders, etc., have also been regarded as abortive forms of epilepsy—*epilepsie larvée* (Trousseau, Féré, Ardin-Delteil, Bellisari, *Ref. méd.*, 1903, etc.), but the proofs adduced are very unsatisfactory. Nor can we admit that enuresis nocturna is a common prodroma of epilepsy. On the other hand epilepsy is sometimes masked by an enuresis which appears at the time of puberty or later. According to Féré, transient deafness may represent the attack ; he also mentions priapism, paroxysmal bulimia or pica, general fatigue and apathy of very short duration (*Rev. de Méd.*, 1903), etc.

A very remarkable variety of the epileptic attack, in which *vasomotor* disturbances are the most prominent symptom, is shown by a case observed and described by P. Meyer and myself. Vasomotor epilepsy is frequently mentioned in the earlier literature. Repeated paroxysmal hypothermia has been found by Ceni and regarded by him as a vasomotor phenomenon. In some cases muscular twitchings of short duration appeared at different sites during the intervals between the paroxysms, consciousness being unaffected. This variety has been described by Russian physicians (Kojewnikoff, Muratoff, Bechterew) under the name of *epilepsia continua* ;

[1] *M. f. P.*, xvii.

[2] *R. of N.*, 1907.

Bruns has also mentioned it. Epilepsia choreica has also been reported (Bechterew, *Z. f. N.*, xii.; Grondone, *Thèse de Lyon*, 1905). Consult the chapter on myoclonia.

Senile epilepsy does not, according to Redlich, essentially differ from true epilepsy, but the amnesia is often incomplete, and conditions of paralysis and disturbance of speech appear frequently to follow the attacks.

Reflex epilepsy in some respects occupies a special position. Injuries at the peripheral parts of the extremities (hand, foot) are the chief cause of the convulsions, but scars in the face, on the skull, and possibly even in the meninges may produce reflex epilepsy. These reflex spasms are usually preceded by an aura, which arises in the injured part of the body and is felt in the form of paræsthesiæ or twitching. These may for a long time be limited to some muscles or to one extremity. The paræsthesiæ and twitching become more extensive in the successive attacks, until consciousness is also lost. In this form the attack is often followed by paralysis of short duration of the extremity first affected, or of the whole side of the body. The cicatrix is usually sensitive and sometimes forms an *epileptogenic zone*, *i.e.* a site at which stimulation (rubbing, a blow, etc.) may provoke an attack, or at which ligature of the extremity may arrest it. An epileptogenic zone is less often found apart from the site of the injury. Some authors think that reflex epilepsy should be classed with hysteria. I have, however, been able to prove in several cases, by means of the well-known criteria, especially by the absence of the pupil reflex during the attack, that we are dealing here with *true epilepsy*. Indeed, I have seen some cases in which the picture was absolutely identical with that of cortical epilepsy, leaving transient hemiplegia with Babinski's sign, etc. The observations of Seeligmüller[1] also show that reflex epilepsy cannot be classified with hysteria, as Binswanger had already recognised. Some of the cases caused by injury to the head undoubtedly belong to the category of cortical epilepsy.

My experience shows that a cicatrix is not always necessary for the production of this disease, but that concussion, traction on a limb, etc., may occasionally give rise to an irritation which is conveyed into the corresponding cortical centre, and which thus acquires an "epileptogenous" condition.

The convulsive symptoms observed after operations for empyema have been interpreted in this way.

The *general condition* of the epileptic is often quite normal. *Signs of degeneration* are frequently present. Malformations of the skull, errors of refraction and other anomalies of development in the eye are found in a great number of cases.

See Schön-Thorey, *A. f. P.*, Bd. xxxix., with regard to ocular malformations in epileptics.

Malformations of the extremities (polydactyly, syndactyly, etc.) are less common. I have been struck in a few cases by the premature and excessive development of the genital organs. Thus in one case of epileptic dementia in a boy of ten, the penis was completely developed and there was a strong growth of hair on the mons veneris; erections had occurred for the last five years, and were said to be distressing in their intensity and persistence. Féré and Lannois describe melanodermia on the thorax.

[1] "Klin. Beitr. zur Reflexepilepsie," Festschrift, Leipzig, 1897. See also Urbantschitsch, *W. kl. W.*, 1906.

The *intelligence* may be absolutely intact. The examples of Cæsar, Napoleon, etc., have often been quoted, but there are no instances of the kind in recent history, although one of the greatest natural philosophers of our times is said to have suffered for thirty years from epileptic attacks. In the majority of cases the mind is affected. Apart from the fact that epileptics are often *excitable*, *suspicious*, and *irascible*, the most common form of mental impairment is *simple* dementia, diminution of the intelligence, and especially of the memory. Hypochondria is occasionally associated with epilepsy. The epilepsy which develops in early childhood is very often combined with imbecility or idiocy, and this mental weakness is co-ordinate with the epilepsy. But in some cases the intelligence which is at first normal may become impaired on account of the prolonged duration of the disease and the accumulation of attacks. Minor epilepsy is not less liable to affect the mind than the major attacks. After a long intermission of the latter, a condition of indolence and mental weakness may develop, which will disappear upon the occurrence of a fresh attack.

As regard the responsibility of the epileptic, the reader is referred to the textbooks of psychiatry and forensic psychiatry, and to Schaffer[1] and others.

Differential Diagnosis.—The difficulty of diagnosis may be great, if the physician only gains his knowledge of the attacks from the incomplete descriptions of the patient and his relatives. One should make it a rule never to diagnose epilepsy upon the ground of a single attack.

A member of the supreme court consulted me on account of neuralgia in the arm, and mentioned incidentally that some ten years previously, after great mental overstrain with night-work, he had had an attack of complete loss of consciousness, with biting of his tongue, convulsions, etc., which had never recurred.

The less the description corresponds to the classic type of epileptic convulsions, the greater is the care required in making a diagnosis. This is specially so as regards simple loss of consciousness. The epileptic attack may be confused with *simple syncope*. The following points serve to differentiate them: Syncope has its origin in anæmia of the brain, caused either by a disturbance of the heart's action, a vascular spasm, a sudden change of position in general anæmia, or some peculiar sensibility to certain mental impressions, *e.g.* the sight of blood. The attack is therefore preceded by some such excitement or by a feeling of faintness arising from the weak action of the heart. The epileptic fit, on the other hand, is almost always sudden and spontaneous in its onset, or follows some definitely characterised aura. In an attack of syncope the heart ceases to beat, or at least the pulse becomes small and weak, but unconsciousness is seldom so complete that the reflexes, those of the pupils in particular, are abolished. A fainting patient not infrequently comes to himself at intervals, and again loses consciousness if he raises himself up. If the unconsciousness is accompanied by convulsions, by passage of urine, or automatic acts, or is followed by a condition of mental disturbance, the diagnosis of epilepsy is practically certain.

I hesitated to give the diagnosis of epilepsy in one case in which a woman, who was quite healthy except for slight vasomotor disorders, became suddenly quite unconscious at a social

[1] *Budap. orvos.*, 1904.

entertainment at the commencement of her period ; her urine and fæces were passed involuntarily, but she showed no symptom of motor irritation, wakened in a quarter of an hour, and thereafter remained quite well. I thought that the cause must be a sudden cerebral anæmia caused by vasomotor disturbances, and as a matter of fact the patient has never had another attack of this kind, though it took place many years ago. I have already described another case in which an attack of anxiety associated with a fixed idea brought on absolute unconsciousness, with passage of urine and fæces. (The remarks under psychasthenic convulsions should also be referred to.) As regards the diagnosis of syncope from minor epilepsy and their possible relations to each other, the reader should refer to Gowers (*Lancet*, 1907).

The diagnosis of the epileptic attack from attacks due to *uræmia*, *lead-poisoning*, and other *toxic* conditions, is founded chiefly upon consideration of the primary disease and the other symptoms of poisoning, etc. Ménière's vertigo may simulate an epileptic attack, but the aural symptoms will indicate the diagnosis in such cases.

According to Gowers (*Brit. Med. Journ.*, 1906), epileptic vertigo differs from the aural form chiefly in its rapidly transient course, the bad effect upon the latter of a change of the position of the body or head, etc. etc.

With regard to the *laughing spasm* which I have described, see p. 673. The diagnosis between *hysterical* and *epileptic* attacks is both exceedingly important and difficult. It was formerly thought that when the convulsion took place under the eyes of the physician, the condition of the *pupillary light reflex* would decide the diagnosis, as it was assumed to be never absent except during an epileptic attack. Recently, however, an increasing number of cases have been published (A. Westphal, Karplus ; see p. 1087), in which this reflex was found to be absent during hysterical spasms, so that some writers go so far as to deny this sign any real diagnostic value. Personally, I have found this absence of the pupil reflex to be exceedingly rare in hysteria, and I still adhere to the view that it indicates the probability that the attack is of an epileptic nature. On the other hand this reflex is occasionally present in rare cases of epilepsy. I have seen this myself in one very interesting case. The patient had for many years suffered from attacks of the nature of narcolepsy or coma, and her physician had, in spite of occasional biting of the tongue, given a diagnosis of hysteria. I was called to the case after the patient had fallen into a state of deep coma, which had already lasted for four to five days. I found her absolutely unconscious, the tendon and corneal reflexes being completely absent ; on the other hand the pupillary light reflex was not only present, but was specially active, and remained so until shortly before death, which took place during this attack. There was no sign whatever of any organic brain disease. A case such as this shows that in some individuals the parts concerned in the production of the pupillary light reflex are endowed with a peculiar power of resistance, and are unaffected by the most profound coma. Other indications pointing to epilepsy are the sudden spontaneous onset of the attack, without preceding mental excitement (although in rare cases an epileptic attack is also brought on by psychic emotion), the epileptic cry, the character of the twitching, which differs from the hysterical tremor in which there is also some trace of voluntary or mental origin (movements of expression). The *arc de cercle*, the salaaming movements, the speaking, crying, and frenzy during the attack, the passionate attitudes, and the spasms of laughing and weeping all point to the hysterical nature of the fit. It is true that crying,

singing, or whistling may, in rare cases, represent the aura or equivalent of an epileptic spasm (Heveroch, Bianchini [1]). The tongue is hardly ever bitten during hysterical fits, but biting of the lips or snapping at those around is very characteristic of hysteria. I have once, however, seen the tongue bitten in hysterical spasm, and other authors (Hoche [2]) have mentioned this as an exceptional occurrence. It is also rare that an hysteric in falling injures himself as severely as an epileptic does. Involuntary micturition and specially defæcation and seminal emission are evidence in favour of epilepsy. According to Charcot urine may be passed during an hysterical attack; (Karplus and I have also seen some such cases). Charcot points out also that hysterical convulsions tend to occur in the evening, and epileptic fits during the night or in the early morning (three to four o'clock). Deep sleep after the attack indicates that it has been epileptic in nature. Babinski has found his toe-sign present during and after an epileptic attack, but never during or after an hysterical spasm. Esmenard [3] has confirmed this. The same statement applies to the Oppenheim sign. The presence of the " feed-reflex " during coma points to the existence of epilepsy.

Should the physician see the patient in the interval between the attacks, his examination should bear upon all these points. The presence of scars on the tongue, which have been acquired during an attack, point to epilepsy. If the convulsions have persisted for some considerable time, the condition of the mental condition is of great importance ; if the intelligence is good, the mind active, and the play of facial expression lively, the case is probably one of hysteria, whilst a stupid expression or marked dementia indicates epilepsy. These criteria should not, however, be considered absolute. Binswanger (and Hoche) have expressed similar views on this point.

I remember upon one occasion going through the ward for convulsive patients in the Charité, in company with the physician in charge, and endeavouring to surmise from the expressions upon the faces of the patients the character of their attacks ; in almost every case my diagnosis was correct.

The *duration of the attack* is also a valuable diagnostic sign. An epileptic attack, exclusive of the sleep which follows it, lasts for a *few minutes* (1 to 3, or 10 at the outside). If the spasm lasts for fifteen minutes to half an hour or longer, the condition is practically always one of hysteria, organic brain disease, or intoxication.

Donath [4] has found choline in the cerebro-spinal fluid obtained by lumbar puncture after epileptic attacks. If this is a constant and pathognomonic condition, it may be used to determine the differential diagnosis in suitable cases.

In doubtful cases one may experiment by inducing an attack by means of *hypnosis* or some such suggestive influence. If this is successful, the case is probably one of hysteria. The relatives usually inform us that the spontaneous attacks resemble those produced in this way. The following points are also noteworthy : if pressure upon the ovaries, or some such device, can arrest or even influence the attack during the stage

[1] *R. n.*, 1904.

[2] " Die Differentialdiagnose zwischen Epilepsie und Hysterie," Berlin, 1902.

[3] *Thèse de Paris*, 1902.

[4] *Orvos. Hetilap.*, 1903 ; *Z. f. physiol. Chem.*, Bd. xxxix. ; *Z. f. N.*, xxvii. See also Mannsfeld (*Hoppe-Seylers Z.*, Bd. xlii. ; *N. C.*, 1905), Mott-Halliburton (*Philos. Trans. London*, 1901).

of commencing loss of consciousness and general convulsions, the hysterical nature of the case is manifest.

It is generally admitted that hysteria and epilepsy may co-exist, the same patient suffering sometimes from hysterical, sometimes from epileptic attacks. As a rule the epilepsy develops first, the hysteria making its appearance later (Falkenberg-Bratz,[1] Hermann [2]). Charcot, followed by Hoche, Falkenberg-Bratz, and others, do not admit the mixed forms of hysterical and epileptic attacks—the so-called hystero-epilepsy, in which Landouzy, Gowers, and others believe—whilst Binswanger, Nonne, and Steffens [3] regard the mixed forms as a rare variety, and Jolly assumes that in exceptional cases hysteria is capable of producing the condition of excitement in the brain which underlies the epileptic spasm.

In the earlier editions of this text-book I maintained the existence of *intermediate* conditions of convulsion, which do not correspond exactly to the type of one or other form. I have specially observed these in individuals of marked nervous heredity who showed signs of mental degeneration. In such cases there are attacks of unconsciousness, with or without convulsions, which tend specially to occur as equivalents of the neurasthenic or psychasthenic conditions of anxiety. I have also occasionally seen a type corresponding to petit mal. Since the publication of the earlier editions, my experience of such cases has become greatly enlarged, and I have reported some of them in my paper upon *psychasthenic convulsions*.[4]

We may here quote two additional cases: Frau H. has suffered from her fifteenth year from attacks of the type of petit mal; the remarkable point about them is that they are apt to occur after any excitement, *e.g.* after every railway journey. General emaciation has developed during the last few years. I prescribed a rest-cure in N., and she gained 20 lbs. in weight. The attacks of unconsciousness entirely disappeared, but a true anthropophobia or fear of society developed.

A woman of 38 has suffered from childhood from migraine and neurasthenia; for the last six years there have been at long intervals attacks of complete unconsciousness, with involuntary micturition and defæcation, which occurred whenever she saw blood, pricked herself, etc.; she has also had agoraphobia.

We may also refer to the case already described of a lawyer with brachial neuralgia and other neurasthenic troubles, who, after excessive mental strain, had a single attack of epilepsy, in which he bit his tongue, etc.

G., a neuro-psychopath, has suffered from conditions of anxiety and imperative ideas. Three times during his life he had an attack of an epileptic character, once in his seventeenth year after great exertion, once in his twenty-third year after sleepless nights, and once at the age of 30 after influenza. In this last attack he was carried home in a state of absolute unconsciousness.

J., aged 8; both his parents were nervous, the father being very eccentric; the patient has always been very excitable and sensitive, and has general tic (in a slight form). Recently he had been compelled by his ambitious father to undergo severe mental strain, to rise at half-past five in the morning, etc.; he became in consequence much run down and suffered from attacks of sudden and increasing heat, sweating, tachycardia, and convulsions; he usually retained his consciousness, but there was sometimes confusion, illusions, etc., at the height of the attack. These convulsions lasted from fifteen minutes to an hour and a half.

For an account of the special character of these convulsive seizures, I would refer the reader to my paper. I have there pointed out that the area involved in these convulsions in epilepsy and in hysteria is not much more extensive than in the cases which I have described there. This is

[1] *A. f. P.*, Bd. xxxviii. [2] *M. f. P.*, xiii. [3] *A. f. P.*, Bd. xxxix. [4] *Journ. f. Psych.*, vi.

abundantly shown by the more recent papers of Friedmann,[1] Heilbronner,[2] and Bratz-Leubuscher,[3] whose observations only partially agree with my own.

Dufour thinks that the fact of convulsions having occurred during early childhood points to their being of an epileptic nature, but I do not regard this as at all a certain indication (see next section).

Amongst the psychical forms of epilepsy, hallucinatory delirium and the "crepuscular" conditions associated with the wandering impulse are specially difficult to diagnose, as these conditions also occur in hysteria. The degree of mental confusion, the violence of the actions, and the causeless, purposeless onset of the attacks characterise the hallucinatory delirium of epilepsy, but in some cases a correct opinion regarding the patient's actions can only be arrived at by considering them along with his convulsive attacks.

The convulsions which occur in children as a symptom of fever should be regarded, not as epilepsy but as eclampsia (*q.v.*).

It may be difficult to decide at once whether the convulsions which occur in individuals with *intestinal worms* belong to true epilepsy or not. The coincidence is usually an accidental one. It is stated that the convulsions produced by tæniæ are of gradual onset, and that the convulsive phase lasts longer than in true epilepsy. The most certain differential sign in all these so-called reflex-spasms is of course the result of treatment.

Nocturnal epilepsy may, for a long time, remain undiscovered, especially if the patient sleeps alone. The following symptoms point to its existence: biting the tongue, spots of blood on the pillows, micturition during sleep, hæmorrhages in the skin and conjunctivæ, bruises of unknown origin, dull headache, dulness and depression in the morning. Irregular breathing, moaning, gurgling noises, etc., indicate an attack during night.

As regards the relationship of epilepsy to migraine, myoclonia, etc., compare the chapters on these subjects.

The differential diagnosis between *true* and *symptomatic* epilepsy cannot always be definitely made. If the epileptic attack is merely a symptom of increased intracranial pressure, as in some cases of brain tumour, other signs of this condition are usually present. It is therefore a matter of the greatest importance that we should in every case of epilepsy look for the signs of an organic brain disease, and above all that we should make an ophthalmoscopic examination. In rare cases tumour may exactly resemble pure epilepsy. We should also remember that epileptic attacks may sometimes exist for years before the development of a brain tumour, so long indeed that one can hardly believe that the interval can correspond to the latency of a neoplasm (see chapter on brain tumour). If the convulsion is of a *cortico-epileptic* nature, the case is very probably not one of true epilepsy. There are, it is true, some rare cases of this disease in which the convulsions are limited to one side of the body, but these do not show the regularity of development and course, nor the progressive character of the cortical epilepsy due to a palpable brain disease, nor the other symptoms associated with such an affection. It may be taken as the rule that the convulsions of true epilepsy become very

[1] "Über die nicht-epileptischen Absenzen oder kurzen narkolept. Anfälle," *Z. f. N.*, xxx.

[2] *Z. f. N.*, xxxi.

[3] *D. m. W.*, 1907, and Bratz, "Das Krankheitsbild der Affektepilepsie," *Ärztl. Sachverst.*, 1907. See also Spiller, *Journ. of Abnorm. Psychol.*, 1907. Westphal (*A. f. P.*, iii.) has also drawn attention to these "epileptic" conditions which do not belong to epilepsy.

rapidly generalised. On the other hand we must admit that the boundary between partial and general epilepsy is not always a clear one. We should specially remember that injuries to the head may, on account of the cicatrices which they leave behind, give rise to a reflex epilepsy, which may be wrongly attributed to a focus in the brain.

Convulsive attacks are frequently observed at the beginning and during the course of *paralytic dementia*. These convulsions are usually unilateral, with or without loss of consciousness. Typical, general attacks of epilepsy are less often observed. The diagnosis should be made from the other signs of the disease.

We must always be on our guard when the epilepsy develops during adult life. It is then usually symptomatic, and may be caused by cerebral tumour, cerebral syphilis, paralytic dementia, chronic nephritis, arteriosclerosis, cysticercus cerebri, etc. It is often also of toxic origin at this age. Recent experience tends more and more to restrict the field of true epilepsy in advanced life, but we are not justified in absolutely denying its occurrence in old age. We find that other "hereditary and congenital" affections may also in rare cases first develop in patients of advanced age.

It is very difficult to determine whether the convulsions which occur in *earliest childhood* are those of true epilepsy or are merely of a symptomatic nature. In any case the convulsions of dentition should not, without further proof, be regarded as epileptic. I believe salaaming spasms should never be regarded as a species of epilepsy, as Féré thought. In the rare cases in which the cerebral paralysis of childhood leaves no disturbance of motility behind it, the accompanying epilepsy is usually characterised by a unilateral onset of the twitching. More careful examination will reveal a diagnostic sign in the tendency, often slight, to associated and athetoid movements, or in a *persistent* Babinski sign. According to Redlich the left-handedness common to epileptics may result from a previous brain disease.

We must finally consider the question of the *simulation* of an epileptic attack, which is sometimes attempted and cleverly carried out. The immobility of the pupils, the pallor at the onset of the attack, the cyanosis, and usually the biting of the tongue, the post-epileptic stupor and confusion, are of course absent. Absence of the reflexes, even of the tendon jerks, is a proof of the genuineness of the attack. Mairet lays great stress upon the examination of the urine, which shows increased secretion of nitrogenous elements and phosphates, and decrease of toxicity after the attack, whilst in recent French literature the much-talked-of "hypotoxicity" of the urine is cited as a characteristic sign (see further on). We must wait for further investigations before admitting the significance of this very doubtful factor.

Course and Prognosis.—The attacks vary even more in frequency than in form. Some epileptics have only an attack once in a year or in several years (Sinkler reports intervals up to twenty-seven years), whilst others have them daily or several times a day. There are many transitions between these two extremes. As a rule the convulsions, especially of major epilepsy, occur once or twice within each month. A succession of attacks, of which there may be twenty or more in the day, hardly ever occurs except in the slight forms of minor epilepsy.

If attacks of major epilepsy follow in rapid succession, without a return of consciousness, the patient is in the dangerous condition known

as *status epilepticus* (état de mal). The temperature usually rises with each attack, and may reach 106° F. In one case which ended fatally, I found a temperature of 107·6° F., whilst Bourneville has seen it rise to 111·2° F. after death. The pulse is small and rapid. The attack may last for several days and end in a condition of great exhaustion or in death.

A variety of status epilepticus has been described in which there are no convulsions, the equivalent being a condition of hallucinatory confusion with fever (Weber) or coma without convulsions (Pick [1]). The Jacksonian type of genuine epilepsy may also lead to a corresponding status epilepticus (Winkler, Landouzy-Siredey, L. Müller [2]). *A series of attacks* with return of consciousness between the convulsions, and normal or slightly increased temperature, should not be regarded as identical with status epilepticus.

The sudden withdrawal of large doses of bromide may produce status epilepticus (Bökelmann [3]). See also the recent articles by Clark-Prout [4]).

Some epileptics suffer only from major, others only from minor attacks, but many from a combination of the two forms.

The attacks come on more frequently during the day than the night. If they occur chiefly or entirely during the night they may remain for a long time undiscovered. It is very remarkable, however, that it is not night, but *sleep* which brings on the attack, as I have had some persons under my care (bakers, printers) whose attacks came on when they were sleeping during the day. They are often associated with the time of menstruation, occurring regularly just before or during the period. They may cease during pregnancy, but this is not the rule.

According to the statistics of Nerrlinger, Fellner, Beckhaus, and others (quoted by H. Curschmann), pregnancy has usually a favourable effect. On the other hand, Echeverria, Fellner, and H. Curschmann (*M. m. W.*, 1904) state that pregnancy may give rise to epilepsy.

Acute febrile diseases often serve to inhibit the convulsions; the intermissions coincide with the pyrexial periods (Marchand-Toulouse), and recovery may even in a few cases be due to this cause (Turnowski,[5] Covéos, etc.). Injuries, operations, and chronic suppuration may also have an inhibiting effect upon the attacks.

The attacks usually come on without any recognisable cause. The patient sometimes attributes them to indigestion, alcoholic excess, physical exertion, coitus, etc. In hospital cases it is often observed that the attacks increase after each leave of absence. In some exceptional cases, mental excitement, fright in particular, have this causal effect, and we are not justified in doubting the epileptic nature of the attack for this reason.

By frightening a child, whom I was treating for infantile spastic hemiplegia and epilepsy, I was able to bring on an epileptic fit. The parents had drawn my attention to this fact, and they also believed that a second counter-fright might inhibit the onset of an attack. See my papers on acoustico-motor discharges in infantile cerebral paralysis.

In a few rare cases, strong sensory stimuli (noises) are said to have caused the fit. A case of Féré's, in which it was brought on by the act of micturition and defæcation, is not one of true epilepsy.

The disease does not endanger life, but a good number of epileptics die early. Status epilepticus is specially apt to prove fatal. One-half of

[1] *W. kl. W.*, 1904.
[2] *Z. f. N.*, xxviii.
[3] *Therap. Monats.*, 1906.
[4] *Amer. Journ. Insan.*, 1904.
[5] *W. m. W.*, 1901. See also Féré, *Journ. méd. de Bruxelles*, 1902.

those in whom this condition develops succumb to it (according to Clark and Prout only 25, or according to Bökelmann [1] about 30 per cent.). Others die from injuries caused by falling during the attack, possibly into the fire. Suffocation may also be fatal, especially if the patient lies face downwards during the convulsive stage. In very rare cases asphyxia, rupture of the heart during the attack, or hæmorrhage from biting the tongue are the causes of death.

Complete recovery is unfortunately unusual, but the number of such cases is greater than is generally assumed. According to the statistics of Habermaas,[2] it occurs in 10 per cent. of all the cases ; Turner is of the same opinion. According to Volland (*Z. f. P.*, Bd. lxv.), out of 4215 epileptics seen in the course of twenty-five years, 245 were cured. Of these 138 could be traced, and in 83 the duration of the recovery was confirmed. I have often found that the relatives of the patients who have consulted me for epilepsy, or some other nervous trouble, had also suffered from convulsions in their youth, which from their description I could only explain as epileptic, but from which they had been absolutely free for five to twenty years. I have also sometimes been told by patients of advanced years who were under my care for neurasthenia, migraine, and other nervous diseases, that they suffered from epileptic fits in childhood until they reached the age of five or eight years, or even later. These facts seem to be of importance, as in our cases of epilepsy we do not consider ourselves justified in speaking of recovery unless the patient has been under our observation for at least five or ten years. Le Duigou [3] has recently collected some interesting observations of recovery which has lasted for many years. Brasset has also reported a very remarkable case in which epilepsy of fifty years' standing began to improve after an apoplectic stroke, and then completely disappeared.

The prognosis is least favourable in the forms of epilepsy which are associated with congenital or acquired mental weakness. The longer the disease has lasted, the less chance is there of recovery. The prognosis has no direct relation to the severity of the various attacks. It seems to me, however, that the prognosis as to recovery is less favourable in minor than in major epilepsy. Turner [4] concludes from his statistics that the prognosis is gravest in the form which commences before the age of ten. Finckh's experience also points to this fact. Alcoholic epilepsy is more frequently cured than true epilepsy, and the form which is due to syphilis is specially amenable to treatment. Reflex epilepsy is also frequently cured, especially if it is not of long standing.

Of the forms due to injury of the skull, recovery can practically only be expected in those corresponding to the type of cortical or reflex epilepsy (see, however, further on).

Pathology, Anatomy, and Pathogenesis.—Post-mortem examination as a rule yields negative results. There are, at all events, no constant changes which can be regarded as the cause of the disease. Thickening of the cranial bones, and thickening and adhesions of the meninges are so seldom found that no importance can be attached to them. Sclerosis of the *cornu ammonis*, to which Meynert refers and to the importance of which Sommer and Bratz [5] call attention—the latter found hypoplasia of the cornu

[1] *Therap. Monats.*, 1906.
[2] *Z. f. P.*, Bd. lviii.
[3] *Progrès méd.*, 1899, and *Thèse de Paris*, 1899.
[4] *Lancet*, 1903 ; *Edin. Med. Journ*, 1904 ; see also Spratling, *N. Y. Med. Journ.*, 1904.
[5] *A. f. P.*, Bd. xxxi.

ammonis—is possibly, as I [1] stated in the discussion on Bratz's lecture, a malformation which is merely a "stigma hereditatis or degenerationis." Bratz seems to have accepted this view, as he also found this alteration without exception in cases of heredity, and Weber has seen it in a number of cases. Holst [2] has had results similar to those of Bratz. It is still uncertain whether the abnormal narrowness of the cerebral vessels and the aorta, which is occasionally mentioned, plays any causal part.

Recent histological investigations of the cerebral cortex have yielded some noteworthy results. Bevan-Lewis, Buchholz, and others had previously found changes which seemed to point to atrophy of the nerve cells, and since then Chaslin [3] in particular has made careful examinations of the epileptic brain. He found proliferation of the glial fibres in the cortex, which are arranged, especially in the external layers, into compact bundles of fibrils; there is therefore a kind of *sclerosis* or gliosis, which may even be apparent to the naked eye. Bleuler [4] has confirmed this change; he found also hypertrophy of the outermost glia layer of the cortex. It is still very doubtful, however, whether these pathological changes are the cause of the epilepsy or the result of the convulsions, as Marinesco [5] and others think.

Kaes (*N. C.*, 1904), to whose investigations we should refer, has not confirmed this marginal gliosis.

Alzheimer (*M. f. P.*, iv.) has lately demonstrated, by exhaustive investigations, that the changes described by Chaslin occur in cases of epilepsy (and dementia). Sailer has also attributed a form of epilepsy associated with dementia to a sclerotic cortical process ("hypertrophic nodular gliosis"). Similar observations have been published by Rosenfeld, Orloff (*A. f. P.*, Bd. xxxviii.), and others. These forms can be easily distinguished from genuine epilepsy, as Voisin has stated.

Onuf (*Journ. Amer. Med. Assoc.*, 1905) describes, in addition to other changes, atrophy of the geniculate body and the thalamus. Turner (*Brit. Med. Journ.*, 1906, and *Journ. Ment. Sc.*, 1907) lays stress on the defective constitution of the nervous system, in particular on the embryonal development of the Betz cells, etc., and he finds changes in the vascular system which he regards as characteristic. We should also refer to the articles by Clark-Prout (*Amer. Journ. Insan.*, 1904), Sala (*Riv. sper. di Freniat.*, 1906), and Buck (*Névraxe*, 1907).

Alzheimer, in his latest report (*N. C.*, 1907), has confirmed and supplemented his earlier statements.

Weber, who found numerous changes, expresses himself with reserve as to their relation to the epilepsy.

In fatal cases of status epilepticus, recent changes are found in the blood-vessels, and hæmorrhages (Weber [6]) which are undoubtedly the result of the convulsions and asphyxia. Clark and Prout ascribe the cell changes also to this cause.

Although pathological anatomy has not given us any definite indications as to the site of the disease, other facts point to its nature and site of origin. The view which formerly prevailed and was specially advocated by Kussmaul and Nothnagel, that the medulla oblongata and the pons were the seat of the disease, has been practically abandoned. The

[1] *A. f. P.*, Bd. xxxi., p. 906. [2] *Psych. en neur. Bladen*, 1903.

[3] "Note sur l'anat. path de l'épil.," *Soc. Biol.*, 1889, and *Arch. de méd. expériment.*, 1891.

[4] *M. m. W.*, 1895.

[5] He thinks at least that this may be regarded as the cause of the glial proliferation, whilst the other changes, such as atrophy of the tangential fibres and chromatolysis of the cells, may be attributed to congenital disposition and the result of the toxic processes. Binswanger failed to find Chaslin's changes in three cases.

[6] *N. C.*, 1898.

experiments of Fritsch and Hitzig, Unverricht, François-Frank and Pitres, etc. (see pp. 677 *et seq.*), have shown that epileptic attacks may be excited from the motor zone of the cortex, and that excision of certain parts of the cortex may arrest the twitching in the corresponding groups of muscles. We know that lesions of the motor area, which are evident to the pathologist, may have the same effect. Although these attacks differ from genuine epilepsy in their unilateral onset, this is due to the fact that the stimulus, whether artificially produced or due to disease, only proceeds from the motor zone of *one* hemisphere. If we assume that epilepsy is caused by a lesion of the motor centres on both sides, the difference in the symptoms would be practically explained. The mental disturbances which follow or usher in the attack, and the weakness of intellect which so often develops in the course of the disease, indicate that the cerebral cortex is the seat of the disease. The fact that in diseases which cause complete interruption of conduction in the motor tract of the internal capsule, the paralysed half of the body may be exempt from the convulsions, is also an indication of its cortical origin. In one interesting case, an epilepsy which had existed for many years disappeared after an attack of apoplexy which had produced hemiplegia. Lately, however, some writers (Binswanger, etc.) have maintained that subcortical centres, the medulla oblongata in particular, are concerned in the production of the convulsions. Ziehen [1] attributes the clonic convulsions to the cortex, and the tonic to subcortical centres, and Ossipow and others agree with him on the ground of their experiments. Binswanger expresses the opinion that in genuine epilepsy the original, exciting cause of the convulsions is a primary lesion of the cortex, but that the discharge produced by this excitement takes effect most rapidly and markedly upon the subcortical motor system. He attributes the fall at the onset of the attack to sudden inhibition of the motor functions (see p. 678), but the loss of consciousness is sufficient to explain this symptom. He bases his view on the occurrence of abortive attacks, in which this inhibitory discharge is the only symptom.

The epileptic attack is regarded as a discharge of accumulated irritation (Hughlings Jackson). Schroeder van der Kolk has compared it to a Leyden jar.

Some authors are inclined, as already mentioned, to think that encephalitis of the motor area, usually in childhood, may produce the changes which cause epilepsy, but this can only apply to a small proportion of the cases. Redlich [2] has recently advocated this view, pointing out that slight symptoms of irritation and paralysis have often a hemiplegic distribution.

The epileptic attack has also been attributed to auto-intoxication. This view, held specially by Voisin, Féré, Bouchard, Péton, Haig,[3] Krainski,[4] Framoti, Caro, Pellegrini, Ceni,[5] Pini, Marchand,[6] Masoin,[7] and others, is based on the observation that after an attack the urine contains many toxic products, and that when introduced into the blood of animals it proves to be more poisonous than urine passed before the attack and in the intervals. During the attack it is hypertoxic, and at other times hypotoxic. It has therefore been inferred that retention of toxic

[1] *A. f. P.*, xvii., xx., and xxi.
[2] *A. f. P.*, Bd. xli.
[3] *Br.*, 1896.
[4] "Obos. psich. neur.," etc., 1896; *N. C.*, 1897; *Z. f. P.*, Bd. liv.
[5] *Riv. speriment. di Fren.*, 1901.
[6] *Rev. de Psych.*, 1902.
[7] *Arch. internat. Pharm.*, 1904.

products in the blood produces auto-intoxication, which gives rise to the epileptic attack (and to gastric disorders, etc.). Certain chemical bodies, such as uric acid (Haig) or ammonium carbonate (Krainski), have been regarded as the cause. Krainski attributes the attacks to retention of uric acid or to its transformation into ammonium carbonate. He thinks that the onset of a convulsion can be predicted from the amount of uric acid secreted. Cololian found that during or shortly after an attack the blood of epileptics, unlike the blood of healthy persons, exercised a toxic effect upon animals. Ceni has specially studied this subject, and upon his theory of the autocytotoxines and antiautocytotoxines in the blood of epileptics he has based a method of treatment, but his teaching and conclusions have been strongly opposed by Sala-Rossi [1] and others. Donath [2] thinks choline and the organic ammonia bases, such as trimethyl-amin, are substances which may cause convulsions, but the experiments of Buzzard-Allen [3] show that choline does not play this part. Voisin insists that congenital disposition is the most important factor in the etiology, autointoxication being merely the exciting cause of the attacks. A transient disorder of the secreting function of the kidneys may possibly be caused by some congenitally morbid condition of the nervous system, which in its turn causes the toxic substances to be retained in the blood. Marchand, who regards gastro-intestinal intoxication as the cause, also premises a congenital predisposition of the nervous system.

In any case, these facts require further consideration, and we are not justified in regarding the theory of autointoxication as firmly established, the more so that the conditions found by Hebold-Bratz, Binswanger, Hoppe,[4] and others as regards the toxicity of the urine are by no means uniform. Binswanger is of opinion that the accumulation of the poisonous substances in the blood is a result of an alteration in the function of the central nerve-cells, due to pathological nervous irritation, and that these toxins are removed during the discharge. The epileptic fit may also influence the metabolism as a motor act, and may allow abnormal products to appear in the urine. Inouye and Saiki [5] state that dextro-lactic acid appears in this way. The fact, apparently proved by Dide and Sacquépée, that after repeated attacks the cerebro-spinal fluid becomes toxic in its action, also points to this conclusion.

In reflex epilepsy the irritation from the cicatrix acts upon the motor zone and produces in it the " epileptic change."

Treatment.—The most important point in the treatment is a thorough examination of the body and very careful consideration of any possible cause. Even factors which bear a doubtful relation to epilepsy, such as tæniæ, diseases of the nasal mucous membrane, *gastric* and *intestinal disorders*, etc., should be taken into consideration, for before we enter upon symptomatic treatment it is our duty to arrest the disease at its source, if this is possible, which it seldom is. One must not think it too trifling a thing to prescribe for the treatment of *tape-worms*, to remove *nasal* or *aural polypi*, or to treat *constipation*. No harm can be done by such treatment, and it is always possible that it may effect a cure. There are, however, very few satisfactory cases of recovery from epilepsy after extraction of a tooth, operation for polypus, removal of foreign bodies from the ear, nose, etc. (see below). Diseases of the genital apparatus

[1] *N. C.*, 1903. [2] *Z. f. N.*, xxxii. [3] *R. of N.*, 1905.
[4] *W. kl. R.*, 1903. [5] *Z. f. physiol. Chem.*, Bd. xxxvii.

should also be considered, although it is very doubtful whether they can give rise to epilepsy. Artificial termination of pregnancy on account of epilepsy is only indicated if the attacks are accumulating to a dangerous extent (Wagner, Redlich [1]). *Injuries* should be specially looked for. If the aura arises from any definite part of the body, the physician should look for scars or other signs of an old injury. If the case can be shown to be one of reflex epilepsy, *removal of the cicatrix* is indicated. This treatment is often not successful, or it produces merely temporary benefit (probably because permanent changes have already developed in the cerebral cortex), but this should not prevent us undertaking the rational method of treatment.

If the illness is due to alcoholic poisoning, the patient should be compelled to *abstinence* and treated in an institution. In addition to this, symptomatic treatment is usually required. If there is any evidence that the convulsions are of a syphilitic nature—the syphilis may not only be acquired, but inherited—treatment with *iodide* and *mercury* is indicated. Cases which these have cured have been reported by Trousseau-Pidoux, Oppenheim, Feinberg, and others. I have seen recovery in a case in which, on account of the character of the epilepsy, which was entirely that of the genuine form, and of the inherited disposition, I hardly expected any result. It is advisable to combine iodide of potassium with the bromides.

The convulsions which occur in gout, uræmia, diabetes, etc. (see Stauder, *M. m. W.*, 1906) call for treatment of the primary disease.

As regards the diet, all *irritating food* must be carefully avoided. Spices, alcoholic liquors, strong coffee, and tea should specially be forbidden. Ziehen thinks bouillon very harmful. As regards the method of Richet and Toulouse, see further on. Alcohol should be strictly forbidden. Cases have been observed in which an epilepsy which had been cured for a long time broke out afresh after a drinking bout (Maisonneuve). All indigestible food should be avoided, and the stomach should never be overloaded. Fluids should be taken in large quantities. The diet should be mixed, but meat should be taken only in small quantities. Milk and vegetables, and easily digested fat foods should be preferred. The statistics of Alt [2] show the great value of vegetarian, and especially of milk diet. Fleury [3] gives very strict directions for the avoidance of auto-intoxication and recommends frequent washing out of the stomach. In nocturnal epilepsy especially, the patient should only have a light meal in the evening, a long time before going to bed.

Exercise in the open air is desirable, but fatigue should be avoided. Strümpell [4] suggests that gymnastics do good by improving muscular tone. Mental overstrain is even more to be avoided than physical. If the patient is still free to *choose* his business, he should avoid any occupation in which the occurrence of an attack would endanger his life. An epileptic should not work upon a scaffolding, near water or upon it, by a stove, etc. If the attacks recur at long intervals and the intelligence is unimpaired, the choice of a calling involving work that is mainly mental cannot be absolutely forbidden. The less demand it makes for intercourse with the outer world, the more suitable is the occupation for the epileptic.

[1] *W. m. W.*, 1906.

[2] *N. C.*, 1902, and *Z. f. P.*, 1904. Voisin-Voisin are opposed to the use of vegetarian diet (*Presse méd.*, 1905).

[3] *Gaz. hebd.*, 1898; *R. n.*, 1898.

[4] *A. f. kl. M.*, Bd. lxxxiv.

A trade which does not expose him to any special risk of injury should be chosen. Farming is eminently suitable. The epileptic should always be as far as possible under careful supervision. Binswanger advises that youthful epileptics should be brought up in a country parsonage. See Weygandt[1] on this matter.

In cases in which the attack is associated with or followed by *transient insanity*, the patient should be *placed in an institution*. The protection of an asylum should also be given to *weak-minded epileptics*, and to persons who suffer from major attacks. A county asylum, under medical supervision, and devoted to the care, treatment, education, and occupation of the epileptic, should be chosen if possible. The town of Berlin has a special asylum for epileptics in Wuhlgarten. There are a number of private institutions also in which great care is given to the treatment of epileptics.

Fischer, Stakemann, and others have given valuable directions as to the principles upon which such institutions should be directed.

Climatic treatment is, so far as my experience goes, of little value, but residence in the country or in the woods may have a beneficial effect upon the nervous system by removing the patient from various harmful factors. Mild *cold-water treatment* may be tried in every case: Strasser has lately spoken greatly in favour of this method. Sea-bathing is, of course, out of the question. I have not found high altitudes to be specially beneficial.

The results of electrical treatment are very doubtful. The usual method is galvanisation of the brain. Galvanisation of the sympathetic may also be tried.

Treatment of the Patient during an Epileptic Attack.—If the aura arises from an extremity, an attempt may be made to arrest the attack by *tightly bandaging* the limb before the aura has extended further. This is successful in some cases. The patient should carry a small belt with holes in it—like a leather garter—and should apply the ligature himself. Forced bending, stretching, or traction upon the limb sometimes inhibits the attack (Bravais). In other cases strong cutaneous irritation is said to have the same effect. Artificial suppression of the convulsion is not always beneficial, however; it may be followed by malaise, a condition of depression and irritability associated with headache and giddiness, for which reason some patients prefer the fit itself. When the aura is of another nature, *inhalation of amylnitrite*, a few drops being sprinkled upon a handkerchief, administration of chloral hydrate in a dose which will produce sleep, or swallowing of a teaspoonful of salt may in rare cases restrain the attack. Should these remedies fail and the attack come on, we can do nothing but see that the patient is laid down so that he is protected as far as possible from injury. The clothes at his neck should be loosened in order that the blood may flow away from the brain as easily as possible, and where there is danger of the patient biting his tongue, we may try, though usually without success, to insert a piece of cork or indiarubber between his teeth.

MacConaghey and Crocq (*R. n.*, 1904) say that the attack can be arrested by laying the patient upon his left side during the tonic stage (?). Lannois (*R. n.*, 1904) has confirmed this to a certain extent.

[1] *Psych. neur. W.*, 1904.

It is not advisable to waken the patient out of a post-epileptic coma or sleep; this usually produces headache and mental depression. *Chloral hydrate*, which may be given per rectum in doses of 3 to 4 g. (45 to 60 grains), is specially good in *status epilepticus*. The earlier it is given the longer is a fatal termination postponed (Landerer). Subcutaneous injections of morphia have very little effect. Injections of hyoscin have been recommended. Wildermuth and Naab advise amyl hydrate in doses of 5 to 8 g. (80 to 120 minims) per diem. J. Hoppe [1] found dormiol (2 to 3 g. (30 to 45 drops)) and proponal (0·3 to 0·4 (4½ to 6 grs.) per rectum) of service. If enemata are not retained, chloroform inhalations may be tried (Bökelmann [2]). I have found this to have a prompt effect in a case of status hemiepilepticus. If these remedies fail, *blood-letting* may be indicated. Pichenot-Castin [3] found lumbar puncture useful in one case. The writers who attribute the convulsions to a large extent to autointoxication recommend the use of aperients, diuretics, lithium carbonate, and intestinal antiseptics.

Drugs should be given in the great majority of cases. When the attacks occur at long intervals of a year or more, I think it advisable and rational not to prescribe drugs. In all the other cases, when the causal indication has been attended to, it is necessary to prescribe something which will cure the convulsions, or at least reduce their number and intensity.

The most effectual drugs are *bromide of potassium* and the other bromides. It is very interesting to see that in almost all the other methods of treatment recommended from year to year, the essential factor is the administration of bromide salts. There is great difference of opinion as to the manner in which this drug should be used and as to its dosage. It is advisable to begin with small doses—3·0 to 5·0 (45 to 80 grs.) per day for an adult—and then, by gradually increasing it, to discover what amount is required for the complete control of the convulsions. It may be necessary to increase the daily dose to 12·0 to 14·0 (approximately 180 to 220 grs.), but this is quite the exception. Smaller doses are naturally suitable for children, but after the age of four, 2 to 4 (30 to 60 grs.) may be given in the day.

According to Féré, with whom we do not agree, the bromide salts can only be expected to do good when they produce lack of energy and drowsiness, and when absence of the pharyngeal reflex shows that they have taken effect (Séguin). Ziehen regards absence of the corneal reflex as a sure sign of bromide poisoning. Gilles de la Tourette waits for persistent dilatation and sluggish reaction of the pupils to show that a sufficient quantity of bromide has been taken. Féré continues to give large doses, and only stops the treatment after the attacks have been in abeyance for a long time. According to Laudenheimer (*N. C.*, 1897), the bromide becomes stored up in the organism.

Fürstner and Binswanger have specially advocated the use of small and medium doses, on an average 75-90 grs. daily, for an adult. If large doses are required, the treatment should be carried out under supervision in a hospital.

The rule is to give the bromide for a long time—one or more years—and only to suspend it if a condition of exhaustion, associated with mental dulness, motor weakness and ataxia, gastric disorders, and enfeebled action

[1] *M. m. W.*, 1902.

[2] *Therap. d. Geg.*, 1906. See also Alt, *M.m. W.*, 1905.

[3] *L'Encéphale*, 1907.

of the heart indicates that bromide poisoning (bromism) has developed. This intoxication is revealed in a few cases by conditions of exaltation. Fürstner and Binswanger are of opinion that bromism need not be feared when the drug is carefully given in medium doses, and that its danger has been greatly overrated. They recommend its being given systematically for a number of years. Bromide acne is seldom so severe as to call for interruption of the treatment, but I have seen a few cases in which this cutaneous affection was a very serious trouble. It is apparently much less apt to develop when bromipin is used, and this is also said to be the case with bromocoll.

Strümpell (*A. f. kl. M.*, 1884) is somewhat sceptical as to the benefit of bromide treatment; he would restrict its use to severe cases, being of opinion that many of the mental disturbances which develop in the later course of the disease are due to chronic bromide poisoning.

I have treated a man of 63 years of age, who had suffered for forty years from epilepsy, and had during all that time taken 4 g. (60 grains) of bromide of potassium, without any ill effect.

The bromide should only be discontinued when the attacks have been absent for a whole year. Voisin and Féré regard bromide as a food with which the epileptic should always be provided. The bromide salts, given in the form of potassium, sodium, or of ammonium salts, or as a mixture of all three salts, should be diluted in large quantities of water. A slightly alkaline water may be used. It may also be given in a cold infusion of valerian root. It is advisable to give the drug in regular doses, two or three times a day. If the attack is likely to occur at a certain hour of day or night, it is advisable to give the *whole dose* four to six hours before the attack may be expected. Some physicians prescribe bromide only every few days in correspondingly large doses.

Some years ago Richet and Toulouse[1] found from experience that preparations of bromide had a much greater effect if at the same time salt is withdrawn from the diet; they recommend therefore a saltless diet and small doses of bromide. The dietary should consist of 1000 g. (35 oz.) of milk, 300 g. (10 oz.) of meat, 300 g. (10 oz.) of potatoes, 200 g. (7 oz.) of flour, 2 eggs, 50 g. (1½ oz.) of sugar, 10 g. (150 grains) of coffee, and 40 g. (1 oz.) of butter—all unsalted; 1 to 2 g. (15 to 30 grains) of bromide of potassium would then be sufficient to control the attacks. Bálint[2] has somewhat modified the diet; he gives 1 to 1½ litres (35 to 50 oz.) of milk, 40 to 50 g. (1 to 1½ oz.) of butter, 3 eggs (without salt), 300 to 400 g. (10 to 14 oz.) of bread and fruit, and prescribes the bromide in the form of bromide of soda in bread (bromopan), of which 2 to 3 g. (30 to 45 grs.) is the dose for an adult. If the patient becomes tired of this diet, vegetables or even meat may be given, but without salt. Other writers, such as Schäfer, Garbini, Hudovernig, Meyer, Zickelbach, Roux, Schnitzer, Eason, Esmenard,[3] Turner,[4] and Dejerine,[5] have reported more or less successful results from this treatment. Helmstädt, Pándy, and others have not found it of use, and they refer to certain dangers from the treatment (the onset of vertigo, weakness, failure of memory, diarrhœa, neuralgia, etc.). Muskens[6] has thoroughly discussed the subject. Alt thinks that the chief value of the treatment lies, not in the absence of salt, but in the simplification of the diet.

[1] *Compt. rend. de l'Acad. des Sciences*, 1899.
[2] *B. k. W.*, 1901, and *N. C.*, 1903.
[3] *Thèse de Paris*, 1902.
[4] *R. of N.*, 1904.
[5] *R. n.*, 1905.
[6] *N. C.*, 1905.

Halmi,[1] and to a certain degree Strümpell, are opposed to the treatment by bromides.

In a few cases bromide treatment entirely suppresses the attacks, but in a greater number it has merely lessened them. Thus, to give only one example, I have seen a patient in whom the attacks, which came on in cycles of fourteen days to four weeks, were kept in check for five years by the use of about 3·0 to 4·0 g. (45 to 60 grains) of bromide a day; in another case the attacks have disappeared under moderate doses of bromide, and the patient has now been cured for about seven years. The fact that intervals of very long duration—two to twenty-seven years, according to Sinkler—may occur between the attacks, even without any treatment, makes it difficult to judge of the results.

In many cases bromide fails or is not well borne, and it is necessary to prescribe some other drug. *Atropin* has the next best effect, at least in a small number of cases. I have seen it do good when the bromides have entirely failed. Here also the dose should at first be a small one, viz. $\frac{1}{3}$ to $\frac{1}{2}$ mg. (say $\frac{1}{200}$ to $\frac{1}{125}$ grain) several times a day for adults, with correspondingly smaller doses, which may be increased after a time, for children. I have used this drug for years, with frequent interruptions. *Belladonna* is also sometimes of service.

An old formula for a pill runs as follows: Extr. bellad. Fol. Bellad. aa 1·0 g. (15½ grains), succ. et extr. liq. q. s. ut. f. pilul. Nr. 100. S. At first one or two, increasing to four to six pills per day.

Potassium bromide may be combined with extract of belladonna, the latter in doses of 0·01 to 0·02 ($\frac{1}{7}$ to $\frac{2}{7}$ grain) H. Jackson gives the latter drug in increasing doses. The combination of potassium bromide with chloral hydrate (in very severe cases), antipyrin, *opium*, etc., has also been recommended. Moeli has seen good results from the combined or alternate use of bromide and atropin. Scopolamin is recommended by Olderoggue.

The bromides are specially apt to fail in attacks of petit mal.

After Gowers had recommended the combination of bromide salts with digitalis, Bechterew[2] advised the combined use of cardiac tonics, and especially of adonis vernalis with the bromides. He gives 3 to 8 tablespoonfuls a day of a mixture containing 2 to 3 g. (30 to 45 grains) adonis vernalis, and 12·0 (185 grains) of potassium bromide, to 200·0 c.c. (7 oz.) of distilled water. By its influence upon the action of the heart and the stimulation of diuresis, this medicine has an alleviating and even a curative effect upon the disease. Cesare also reports good results, and I can say from personal experience that this mixture is in some cases more successful than simple bromide treatment. Bechterew has occasionally added codeine to his mixture.

Radix artemisiæ and the *zinc salts*, particularly the oxide and valerianate of zinc, have merely a historic interest, though Gowers has again advised them. With this disease we are often in a position of seeking desperately for remedies, and are therefore forced to fall back upon the old prescriptions. Cocculus Indicus and sodium nitrite may be referred to in this connection.

Among the substitutes for potassium bromide, *borax* may be mentioned: the dose at first should be 0·5 to 1·0 (7·75 to 15·5 grains), later 4·0

[1] Abs. in *N. C.*, 1904.

[2] *N. C.*, 1894 and 1898.

to 5·0 (60 to 75 grains), taken after meals, *e.g.* in the form of sodium biborate, 10·0 (150 grains), aq. dest. 150·0 (5 oz.), up to two spoonfuls three times a day. When this is used for a long time, cutaneous eruptions of an eczematous nature and conjunctivitis may develop, which, like bromide eczema, should be treated with arsenic. Nitroglycerine, prescribed in alcoholic solution in doses of $\frac{1}{10}$ to $\frac{1}{2}$ of a mg. ($\frac{1}{500}$ to $\frac{1}{100}$ grain) is hardly of any use in the treatment of epilepsy.

Among the other bromide preparations, *ethylen bromide* should be specially mentioned.

Rp. Ethylen bromide 5·0 (75 grains), ad emuls. oleos. 1000·0 (35 oz.), ol. menth. pip. gutt. 2. Adults may take thirty drops 2 to 3 times a day in half a glass of sweetened water, increasing the dose every third day by ten drops, until 70 drops or a teaspoonful are being taken. Children of eight to ten years old should begin with 10 to 20 drops. It may also be given in gelatine capsules containing 3 drops, and 6 drops of oil of sweet almonds, 2 to 4 capsules being taken several times a day.

Strontium bromide, strongly approved of, *e.g.* by Bennion, calcium bromide, gold bromide, zinc bromide, bromæthylform, bromalin, bromeigone, brompepton, and bromokoll have also been recommended. Bromipin (the bromide salt combined with oil of sesame), one teaspoonful equalling 2 g. (30 grains) of potassium bromide, has gained special favour during the last few years ; it is prescribed in tablespoonful doses. This preparation has a very bad taste, but is efficacious, as a rule causes no acne, and is also valuable from its nutritive properties (Gessler, Leubuscher, Rohrmann, Lorenz). I have found it useful in a number of cases, notably in four patients, whose attacks have quite ceased for a number of years. It may be had in the form of tablets, and may be given per rectum (Kohte).

Hasle and Bourneville regard monobromated camphor in doses of 0·1 to 0·2 (1·5 to 3 grains) as specially good for attacks of giddiness in epilepsy. Lithium carbonate (0·5 to 3·0 per diem (8 to 45 grains)) is prescribed by Krainski, on the ground of his theory already mentioned. Amylene hydrate is also given in aqueous solution (one to ten), in doses of 2·0 to 4·0 (0·5 to 1 dr.), but it is not to be depended upon.

Parnassia palustris is given by Moravcsik (*N. C.*, 1903) in combination with paraldehyde, two coffee-spoonfuls of the herb put into half a litre (17·5 oz.) of hot water for 15 minutes, then filtered, and 2 g. (half a drachm) paraldehyde added ; this is given in three portions within 24 hours.

If the epilepsy has developed after malarial infection, quinine should be tried.

The *combination of opium and bromide* (Flechsig [1]) has gained in favour in the last few years, the opium being at first given alone, and then the bromide, also by itself. The extract of opium is at first given in doses of 0·05 (0·75 grain) two to three times a day, and gradually increased to 1·0 (15 grains) per diem (0·25 to 0·35 (4 to 5 grains) in each dose). For children of nine to twelve, the largest daily dose is 0·4 g. (6 grains). After about six weeks the opium is suddenly stopped and bromide given in large doses (about 7·5 (say 116 grains) per diem). After the bromide has been taken for a couple of months the dose is reduced to 2·0 (30 grains) per diem. The opium seems to act as a preparation and to increase the effect of the

[1] *N. C.*, 1893 and 1897.

bromide. Ziehen[1] emphasises the necessity for avoiding not only alcohol, coffee, and tea, but all condiments and extractives, bouillon in particular. Whilst the opinion of Binswanger-Warda, Hascovec, Biro, Kellner,[2] Seige,[3] and others is in favour of this treatment, it may be assumed from the experience of others (Bratz, Schroeder, Linke, Donath, Séglas-Heitz, Gowers, Russell) and from the evidence of the published statistics as a whole, that it hardly ever leads to prolonged recovery, and only in a small percentage of cases to real improvement, whilst the method is not without danger and may have a bad effect upon the mental condition. In any case it should only be carried out in hospital and under the strictest supervision.

Opocerebrin, which Lion regards as a panacea, given in doses of 0·2 to 0·3 (3 to 4·5 grs.) or even 2·0 (30 grs.) per day, has proved a failure when used by other physicians, myself included.

The serum treatment of epilepsy inaugurated by Ceni (*C. f. Gr.*, 1902; *N. C.*, 1903; *C. f. N.*, 1905) has been opposed by Roncoroni (*A. di P.*, xxiii.), Sala-Rossi (*N. C.*, 1903), Catola (*Riv. di Patol. nerv. e ment.*, 1903), and especially by Schuckmann (*M. f. P.*, xix.), whilst the observations of Wende, Mazzei (*Rif. med.*, 1904), Gerhartz (*N. C.*, 1904) seem to demand further trial of this method.

Occasional blood-letting is justified if there are symptoms of cerebral congestion or hyperæmia.

Surgical treatment is hardly ever necessary except in *reflex-* and *traumatic epilepsy*. The observation that an injury is capable of suppressing the fits for a certain period has led to the artificial production and maintenance of suppuration. Thus, in a few cases, I have seen Westphal, during the latter years of his life, introduce a seton into the neck. This method is now used by very few physicians. Féré applies a button cautery to the head. When an attack of true or partial epilepsy is ushered in by an aura from a certain part of the body, a *blister* may be applied for a long time above this site. Bravais, Hirt, and Buzzard have found this method successful.

Reflex epilepsy calls for excision of cicatrices, their detachment from the bones, the freeing of a nerve from a callus, the removal of a foreign body, a tumour, etc. When this fails, it is advisable to stretch the corresponding nerve. This has led to recovery in a case under my care, in which no scar could be found on the extremity from which the aura originated. Pick, Hitzig, Bergmann, Winkler,[4] Seeligmüller, and others report successful cases of this kind. Unfortunately permanent recovery can only be expected in some of these cases.

In a few rare cases operations on the uterus (Toulouse-Marchand, Elseworth, and others), removal of foreign bodies and polypi from the ear or nose (recent cases by Grosskopf,[5] Woakes,[6] Lang[7]), evacuation of an empyema of the antrum of Highmore (E. Meyer), enucleation of a blind eye (Echeverria, Hodgdon), expulsion of intestinal worms, etc., have had a curative effect, but there is some doubt as to the genuineness of the epilepsy in the majority of these cases.

The epilepsy caused by injuries to the skull often calls for surgical

[1] *Therap. Monatsch.*, 1898.
[2] *D. m. W.*, 1903, and *M. m. W.*, 1906.
[3] *M. f. P.*, xxii., Erg.-H.; see bibliography here.
[4] "L'intervention chirur. dans les épilepsies," Paris, 1897.
[5] *A. f. Laryngol.*, xiii.
[6] *Lancet*, 1902.
[7] *Budap. orvos.*, 1904. See also Plavec (*N. C.*, 1906), Knapp (*M. f. P.*, xv.), Russell (*Glasgow Med. Journ.*, 1904), and in particular Frey and Fuchs (*Obersteiner*, xiii.).

intervention. In most cases the irritation arises from a *cortical lesion* (adhesion of the cortical surface with the meninges, scars, cysts, plates in the cortex, splinters of bone, etc.), or from the pressure exerted upon the cortex by a depressed fragment of bone. The attacks have not always, however, the character of cortical epilepsy. It should be remembered that traumata, *e.g.* from spent shots, which do not injure the bones of the skull on account of their elasticity, may injure the cortex.

In cases of injury to the head, trephining may be beneficial: (1) if the attacks have the character of cortical epilepsy; (2) if the cicatrix lies above the motor zone. According to the advice of Horsley and Bergmann—with whom Sachs-Gerster,[1] Rasumowsky,[2] F. Krause,[3] and others agree—it is not sufficient to remove the bones, the cicatricial-meningeal tissue, the cyst, etc., but the cortical centre from which the irritation arises should also be excised. This is necessary even when there is a negative condition; the superficial layers of the cortex to the depth of about 5 mm. should be removed. This procedure is naturally followed by paralysis of the corresponding area, but this tends to disappear within a short time, as I have repeatedly found. Others have expressed themselves as opposed to this method. Among thirty-one cases of traumatic cortical epilepsy treated in this way, Raymond found nine failures, nine improvements, and thirteen with apparent recovery; only three of these, however, have been under observation more than three years after operation. Others go still further, and would operate in this way if the attacks take the form of Jacksonian spasms, even when there has been no injury to the head and no scar can be found. The corresponding centre is then determined by electrical stimulation. See Krause for details of the method. An interesting case of non-traumatic epilepsy in which the localised motor aura led to surgical treatment and marked improvement is reported by Eulenburg-Rinne.[4] The communications of Friedrich,[5] F. Krause, and Auerbach-Brodnitz[6] also deserve to be studied. It should be borne in mind that although in partial epilepsy the cortex may have a normal appearance, definite changes may be revealed by microscopic examination (Coën, Gieson, Oppenheim, Collins), but that other cases occur in which no anatomical changes can be discovered (Raymond, Auerbach-Brodnitz).

On the whole, the reports of the results of surgical treatment of traumatic epilepsy are not very encouraging. A large number of cases have certainly been described in which recovery has followed operation in traumatic cortical epilepsy and even in a few cases of general epilepsy of traumatic origin (Horsley, Keen, Lloyd-Deaver, Taylor, Braun, Gussenbauer, Sachs-Gerster, Bremer, Nawratil, Hochenegg, Kocher, Schede, Krause, Kümmel, M'Cosh, Championière, Engelhart, Donath,[7] Rasumowsky, Schulze-Berger,[8] Putnam,[9] Friedrich,[10] Bourneville,[11] Engelhardt, Delbet,[12] and others).

If we consider the communications and statistical reports of Bergmann, Chipault, Starr, Braun, Graf,[13] Höfer, Thouvenet, Eyk, Sachs-Gerster, Häckel, Mathiolus, Pilcz, Putnam, Doran

[1] *D. m. W.*, 1896. [2] *A. f. kl. Chir.*, Bd. lxvii.
[3] "Die deutsche Klinik," etc., and *B. k. W.*, 1905.
[4] *D. m. W.*, 1906. [5] *Verhandl. d. deutsch. Ges. f. Chir.*, 1905.
[6] *M. m. W.*, 1907. See also Kotzenberg, *Beitr. zur. kl. Chir.*, Bd. lv.
[7] *W. kl. W.*, 1903. [8] *A. f. kl. Chir.*, Bd. lxxii.
[9] *Trans. Amer. Med. Assoc.*, 1901. [10] *Therap. d. Geg.*, 1905.
[11] *R. n.*, 1902. [12] *R. n.*, 1904. [13] *A. f. kl. Chir.*, Bd. lvi.

(*Alb. Med. Journ.*, 1902), Fraenkel (*W. kl. W.*, 1905), Nast-Kolb (*Z. f. Chir.*, Bd. lxxiii.), Bullard (*Bost. Med. and Surg. Hosp.*, 1905), etc., we find that the failures predominate, and especially that, as Bergmann first pointed out, even in the favourable cases, the beneficial effect of the treatment has usually been merely a temporary one. The paper by Graf upon the statistics of traumatic Jacksonian epilepsy is particularly comprehensive, thorough, and critical. It comprises 146 cases. In 71 of these trephining was performed with or without opening of the dura, but no incision was made upon the brain; in the other 75 the operation extended to the meninges and the brain. In 56 of these cases there was removal of a fragment of bone from the brain, incision or excision of a cyst, removal of a cicatrix, etc.; in 19 the corresponding cortical centre, which was determined by faradic stimulation, was excised. The cicatrix on the skull corresponded in 92 cases to the cortical centre from which the attack originated. Of the 146 cases of trephining, 6·1 per cent. were fatal (and there were other ill-effects of the operation). Recoveries which lasted more than six months after the bony depression was removed, the cicatrix or cyst excised, etc., or after extirpation of the apparently unaltered cortical centre, took place in 35 cases, *i.e.* in 23·9 per cent., improvement in 22 cases, *i.e.* 15·1 per cent., and failure in 36, *i.e.* 27·6 per cent. In 53 cases the period of observation was too short to enable them to be included in the statistics. Recovery of over three years' duration was noted in at the most 6·5 per cent.

Sachs and Gerster state that results are most likely to be satisfactory in fresh cases, in which the injury dates at most one to three years back. Emerich-Navratil [1] have expressed the same opinion, but recovery has been obtained after intervals of much longer duration (Horsley). Indeed, in a case of Larrey's, removal of a splinter of bone after thirty years is said to have been followed by recovery. In any case it is highly advisable to operate as soon as possible.

Failures in operation are partly due to the fact that the "epileptogenous change" is frequently not limited to the site of the lesion, but extends to other regions of the cortex (Jolly [2]), and also that the operation may give rise to certain changes and secondary conditions. Kocher regards the increase of intracranial pressure (*q.v.*) as a special cause of failure.

We cannot unfortunately yet lay down any clear indications for operative treatment. Bergmann would not extend it to cases of traumatic origin which assume the form of classical epileptic attacks, nor is it justified in cases of mental equivalents of epilepsy, even when these are of traumatic origin (the operation was said to be successful in a case of this kind reported by Pilcz, and Bonhöffer describes a similar interesting observation). Other surgeons, such as Tissot, Chipault [3] M'Cosh, and in particular Kocher, extend the indications much more widely. As we gather from the publications of Kocher and his pupils (Berczowski,[4] Ito,[5] Schär), they consider that epilepsy falls chiefly under the domain of surgery, at least in so far as the cases which have not responded to other treatment. Kocher [6] is of opinion that a local and general increase of the intracranial pressure is the actual cause of epilepsy; he therefore attaches the greatest importance to diminution of this pressure by opening the dura, draining (after Mikulicz), and preventing bony closure of the trephine opening. He proves by statistics that his results have been much more satisfactory since he has employed this method. His data have, however, not been generally accepted, and his theory has proved untenable, as increase of intracanial pressure is not the cause, but is an accessory symptom and a result of the epileptic attack (Nawratzki and Arndt,[7]

[1] *Budap. orvos.*, 1905. [2] *Charité-Annalen*, 1895.
[3] *Gaz. des. hôp.*, 1902; also "Traité chir. operat. du syst. nerv."
[4] *Z. f. Chir.*, Bd. liii., and "Trav. de Neurol. chirur.," 1899. [5] *Z. f. Chir.*, Bd. lii.
[6] *Z. f. Chir.*, Bd. xxxvi.; *Verh. f. Deutsch. Ges. f. Chir.*, 1899; *N. C.*, 1899.
[7] *N. C.*, 1899.

Ormea ;[1] compare also Subsol,[2] and the paper by Fedorow). Bier's[3] statement that artificial increase of intracranial pressure produced by his "congestive bandaging," does not give rise to attacks, is also worthy of notice in this connection. Even Horsley in his latest publications has greatly restricted the scope of indications for operation. From the literature and our own experience, we may draw the following general conclusions :—

1. Operative treatment of true epilepsy of non-traumatic origin is not justified. I adhere to this opinion, although I am aware that some surgeons have of late years extended the indications beyond these cases.

2. Operative treatment of Jacksonian epilepsy of non-traumatic origin is admissible under certain conditions, *e.g.* if an operable cortical affection (tumour, cyst, abscess, etc.) is probably present. Should this not be so, the prospects of successful operation are very slight.

3. Operation is indicated in cases of cortical epilepsy following an injury, especially if the cicatrix practically corresponds to the cerebral motor area. If it lies at a distance from the Rolandic area, the site for operation should be chosen, not at the cicatrix, but at the point indicated by the form of the attack.

4. In these cases all the lesions (cutaneous scars, depressed or thickened parts of bone, splinters of bone, meningeal scars, cysts, etc.) which cause intracranial pressure should be removed, and it is advisable also to excise the corresponding cortical centre itself.

5. In general traumatic epilepsy, operation is justified under the following conditions :—

(*a*) If the scar on the skull forms an epileptogenous zone, or if there are other indications that the case is possibly one of reflex epilepsy. In such cases the cicatriced soft parts should first be loosened from the bone and removed, and the result of this measure should be watched. If it proves to be insufficient, the endocranial cicatrix should be attacked ; (*b*) if there are bone depression and symptoms of meningeal irritation (headache, sensitiveness of the scar to percussion, etc.), which point to a local process of irritation ; (*c*) if the fit, though corresponding to the type of genuine epilepsy, is preceded by an aura of a definite character or followed by some sign of paralysis which points to the probability of a circumscribed cortical lesion (Binswanger).

6. Operation should be followed by prolonged and systematic medicinal and dietetic treatment.

Ligature of the carotid and vertebral arteries need hardly be considered as a method of treating epilepsy.

Removal of the superior sympathetic ganglion, first carried out by Alexander and then almost entirely forgotten, has recently been revived and recommended as a method of treatment. Chipault[4] and Jonnesco[5] in particular have used unilateral and bilateral resection of the superior sympathetic ganglia or of the cervical sympathetic along with these ganglia in a number of cases, and they regard it as a valuable method, as in not a few cases which had resisted treatment of other kinds, it has led

[1] *Arch. ital. de Biol.*, Bd. xxxviii. [2] *Thèse de Paris*, 1903. [3] *Mitt. aus Grenzgeb.*, vii.

[4] *Gaz. hebd.*, 1898, and "Trav. de Neurol. chir.," 1901 (*R. n.*, 1902). See also Vidal (abs. *R. n.*, 1904.)

[5] *Arch. Sc. de Méd.*, 1898 and 1899.

to recovery or marked improvement. From the cases published the operation does not seem to be really dangerous to life, but on the other hand it is very improbable that it actually has the curative effect attributed to it. In any case French neurologists, who have had an opportunity of observing cases upon which Chipault and others have operated, notably Dejerine and Lannois-Jaboulay,[1] deny emphatically that this measure cures epilepsy (the cases which recovered being actually hysterical), and refuse to consider that an operation which may have such ill effects can be justified. Jaboulay[2] has subsequently, it is true, reported one case with a favourable result. Donath, Bramann, Braun[3] (who performed the operation in nine cases with no result), Postempski-Sciamanna, Pilcz, Mariani, and others have expressed the same opinion.

We cannot discuss treatment by means of injection of normal nerve substance and other extraordinary methods, such as artificial infection with pneumonia, etc. (Turnowsky).

Eclampsia

1. Infantile Eclampsia

General convulsions frequently occur in early childhood. Although these usually correspond to the type of an epileptic attack, they may occur under conditions and show peculiarities of course and prognosis which make it necessary to distinguish them from epilepsy. In this sense we are justified in grouping the general convulsions of childhood under the term *infantile eclampsia*, and in giving them as far as possible separate consideration.

We must regard the constitution of the child's brain as the reason why stimuli, which would not elicit convulsions in the adult, are sufficient to produce general convulsions with or without loss of consciousness. We speak of an *increase of the general reflex excitability*. I agree with Soltmann, who uses the term *physiological spasmophilia* of childhood, in the opinion that *defective development of the inhibitory apparatus* is the cause why stimuli of many different kinds give rise to infantile convulsions. A certain, although superficial analogy therefore exists between the brain of a child and of a hysteric.

The term spasmophilia is preferred to that of eclampsia by Heubner, Escherich, Finkelstein, and Thiemich, who refer to its relation to tetany (*Med. Klinik*, 1906; *C. f. N.*, 1906); but Thiemich would include under this heading only cases which are characterised by galvanic inexcitability of the nerves.

It is not uncommon for *acute infective diseases* in children to commence with general convulsions. This is the case also with acute inflammatory affections of the middle-ear. In a few cases (Tumpowski[4]) convulsions have followed vaccination for smallpox.

Gastric and intestinal diseases are a frequent cause of infantile eclampsia. Intestinal catarrh, *severe diarrhœa*, or even simple indigestion (overloading of the stomach) may in early childhood give rise to convulsions. The causal effect of intestinal worms, which is supported by earlier experience and by recent observations (Naab, Festa), has been disputed by many writers, such as D'Espine and Bong.[5]

[1] *Rev. de Méd.*, 1899.
[2] *Lyon méd.*, 1902.
[3] *B. k. W.*, 1896, and *Z. f. Chir.*, Bd. xlviii.
[4] *Gaz. lek.*, 1902; *N. C.*, 1903.
[5] *Ann. de méd. et chir. infant.*, 1902.

Dentition, in particular difficulty in cutting a tooth, is a recognised cause of eclampsia.

Injuries and mental emotions (fright) must also be regarded as causes.

Rickets undoubtedly helps to bring on convulsions. The relation of eclampsia to tetany and laryngospasm will be discussed elsewhere. Status lymphaticus has also been regarded as a cause. Uræmic convulsions and those due to direct poisoning (alcohol, opium, atropin, santonin, etc.) must be distinguished from eclampsia.

The neuropathic and toxicopathic heredity—alcoholism or lead poisoning in the parents—may lay the foundation of this disease (D'Espine).

Toxic, reflex, and *psychic* factors are therefore concerned in the production of infantile eclampsia. In many cases the underlying cause cannot be discovered. It is not definitely known how rickets leads to convulsions. The disturbance of the general nutrition, the influence of chemical bodies, and the softness of the skull bones may all be contributing factors.

The eclamptic attacks usually resemble those of epilepsy, but the development and succession of the symptoms is different, and the attacks have a tendency to occur in series. There is often also a repeated alternation between general tonic and clonic twitchings during the attack, or the clonic stage may precede the tonic. Ausset notes the absence of the initial cry. There may be slight, very transient convulsions accompanied by a cry, but not by any loss of consciousness. Restlessness, irritability, hyperæsthesia of the special senses, and partial convulsions may precede the attack.

It is very difficult to classify the different types of eclampsia. In some cases the convulsions disappear after the cause is relieved; the illness consists only of a few attacks, or it may last for a couple of days or weeks and then entirely disappear. In other cases it may last for months or for a year and more, and then completely pass away. If the patients are kept under further observation, it will be found that some remain quite healthy, whilst others after many years become victims of *epilepsy*, *hysteria*,[1] *neurasthenia*, or *psychasthenia*.

It is also certain that convulsive attacks in early childhood often indicate the existence of *epilepsy*. I think it is proved also by a few cases of my own that *hysterical attacks* may occur in *infancy*. Finally, we should bear in mind that infantile eclampsia is in many, perhaps the majority of cases, a *transient disease* peculiar to childhood, which certainly endangers life, but when once over has no effect upon the future health, except that it may be an early manifestation of the neuropathic diathesis.

The investigations of Thiemich are specially interesting in this respect. He was able to prove that a comparatively large number of these children showed signs of weakmindedness or feeble intelligence, or symptoms of the neuropathic diathesis, *e.g.* disturbances of speech, whilst about a third were in every way normal. These investigations are not, however, adapted to show the relations of infantile eclampsia to epilepsy, as the children were only observed until the age of seven to twelve, and epilepsy tends to develop later. But it was very remarkable that none of these

[1] The fact, which I mentioned in the first edition of this textbook, that eclampsia is sometimes the first sign and prodroma of hysteria, has been subsequently confirmed by several writers, *e.g.* Bruns, Fürstner, Bézy, etc.

children suffered from epilepsy. Thiemich has ascertained that spasmophilia is sometimes a hereditary, family disease.

Organic diseases of the child's brain often give rise to convulsions, but, if we except meningitis, which can usually be recognised at an early stage from other symptoms, these convulsions are generally unilateral. Finkelstein certainly remarks, and rightly so, that even such convulsions do not necessarily imply an organic disease. We are hardly justified, according to his experience, in regarding convulsions which have lasted for more than twelve hours as due to eclampsia; they should suggest meningitis, in particular the serous form and the bacillary "meningitis sine meningitide." On the other hand coma, lasting even for twenty-four hours, does not exclude the possibility of eclampsia. Lumbar puncture may, perhaps, aid one in making the differential diagnosis.

The *prognosis* of eclampsia is favourable in cases with single attacks, but becomes more grave if they persist for a long time and occur very frequently. Severe and rapidly successive attacks are specially dangerous to weakly children. There is every fear that the case is one of epilepsy if the convulsions persist after the toxin is removed, or if no cause can be detected. If the child remains conscious throughout the attack, which in other respects is of an hysterical type, the prognosis as to life is favourable. One must, however, be careful not to confuse these convulsions with those caused by an organic brain disease.

Treatment.—The first necessity is to cure the primary disease. Treatment of gastric catarrh or simple indigestion by the administration of an emetic or an aperient, the evacuation of thread-worms, etc., may rapidly have this effect. If difficult dentition is the cause, lancing the gums may be a prompt remedy. Rickets should be treated in the usual way.

Potassium bromide should be given for the attack itself. In very small children, 0·1 to 0·2 g. (1·5 to 3 grs.) should be given several times a day, this dose being doubled or trebled in children of two to three years of age. Should this fail and the attacks be so frequent or severe as to endanger the child's life, it may be advisable to give small doses of *chloral hydrate.* It may be necessary to give inhalations of *chloroform* if the attacks follow in rapid succession and the child's life be in danger. *Wet packs* and hot or lukewarm baths have been recommended, and cold douches are specially good in hysterical convulsions. Counter-irritation, in the form of mustard plasters to the neck and extremities, has sometimes a good effect. Lumbar puncture should only be tried in severe cases.

2. Eclampsia Gravidarum

Recent literature in the resumé of Schnürer, *C. f. Gr.*, 1903; also in Seydel, *D. m. W.*, 1904; Glitsch, *D. m. W.*, 1904; Esch, *Z. f. Geburt.*, Bd. lviii.

These spasms occur in the latter part of pregnancy, during parturition, or in the puerperium. Young primiparæ are chiefly affected, and the case is usually one of head presentation.

The *origin* and *nature* of the eclampsia of pregnancy is still not fully explained, in spite of very numerous investigations, opinions, and theories.

The oldest theory, that of *uræmia*, specially maintained by Halbertsma and Kundrat, starts from the assumption that the disease is caused by

the mechanical factor of compression of the ureters. Although this theory can no longer be upheld, many writers still believe the disease to have a *renal* origin. This is supported by the almost constant discovery of renal disease in the post-mortem examinations (Nagel, Schmorl, etc.), the great frequency of albuminuria and the occasional occurrence of other nephritic symptoms (retinitis albuminurica, amaurosis, œdema, etc.). But albuminuria is sometimes absent or very slight; microscopical examination of the urine often reveals no changes (Olshausen), etc.; and it is certain that many women suffering from renal disease pass through pregnancy without showing any symptoms of eclampsia.

Many obstetricians hold the view that insufficiency of the kidneys is merely an accessory factor, which renders elimination of the virus producing the convulsions difficult or impossible.

Others regard the renal affection as a mere secondary change, due to the morbid agent, as corresponding processes of degeneration are also often found in the *liver*.

The theory of insufficiency of the parathyroids, held by Vassale (abs. *N. C.*, 1906), cannot be discussed here.

The theory of an *infective* and in particular of a *toxic* origin of eclampsia gravidarum has gradually gained more and more favour. The assumption of a specific bacillus (Schreiber, Neumann, Haegle, Gerdes, etc.) has indeed been strongly disputed (Hofmeister, Olshausen), but some writers, Stroganoff [1] in particular, adhere to the view of an acute infective disease. The hypothesis that the condition is due to toxic products which find their way from the placenta or the fœtus into the maternal circulation (Fehling, Hoeven, Czempin, Weichardt-Piltz,[2] etc.) is better founded; it seems quite probable that this poisoning would be more apt to occur if the functions of the kidneys were impaired. Such a combination of causes is assumed, *e.g.* by Ahlfeld,[3] Kaltenbach, and others.

Zweifel (*A. f. Gyn.*, Bd. lxxii.) believes that the results of examination of the urine show that the albumin is insufficiently oxydised.

Finally, it is possible that an increased predisposition of the nervous system caused by pregnancy or the puerperium may be a factor; thus L. Zuntz and Blumreich [4] have found that the motor centres of pregnant animals showed increased sensitiveness for creatin.

Other anatomo-pathological changes include embolisms of compound granule cells and occasional brain hæmorrhages. The former are of no real significance, and the latter may be regarded as an accessory or secondary symptom of the disease, or as a complication. According to recent investigations (Lubarsch, Schmorl, Seydel), focal degenerations in almost all the organs which are necessary to life and multiple thromboses are the typical conditions found on section. As regards the changes in the central nervous system see Pollak (*Obersteiner*, xiii.).

The spasms may be preceded by restlessness, headache, vertigo, and tenderness of the stomach to pressure (Dührssen); they are sometimes ushered in by a kind of aura (Olshausen). The attacks resemble those of epilepsy, but have a greater tendency to occur in a series. In severe cases the condition is allied to that of status epilepticus. The temperature rises with each attack (Schauta), and may become very high.

[1] *M. f. Geburtsh.*, Bd. xlii.
[2] *D. m. W.*, 1906.
[3] *Z. f. prakt. Ärzte*, 1901.
[4] *A. f. Gynäk.*, Bd. xlv.

Amaurosis (Knapp), much less often hemianopsia (Pick, Knapp[1]), paresis of the extremities (Schwab, Büttner), and mental disturbances may be present for a time after the attack. Among 200 cases of eclampsia, there were 7 in which puerperal mania developed (Dührssen).

Eclampsia without eclamptic fits, and psychoses which replace them (Donkin), have been described, but these are very uncertain conditions. In a few cases eclamptic attacks have been observed in mother and child.

The disease is a grave one, and may prove fatal. Death occurs in about 25 per cent. of the cases (Olshausen), during coma or an apoplectic attack, or from sepsis. Fat embolisms have often been found in the lungs (Virchow). The mortality has become less within recent years. Parturition has a favourable effect.

Treatment has to fulfil three indications: (1) to hasten delivery, (2) to diminish the excitability of the brain, and (3) to accelerate the elimination of the toxin which is probably circulating in the organism. With regard to the first indication, the views of obstetricians differ greatly; some, *e.g.* Fehling, Löhlein, and Fritsch, would, even when the os is not dilated, induce parturition by dilatation by means of indiarubber bags (Wyder), by incision of the vagina and cervix (Dührssen), or by Cæsarean section; others object to forcible hastening of delivery, a view which has gained ground of late. The more recent views on this question and the various methods of treatment have been collected by Glitsch and by Bumm.[2] In order to assist in the elimination of the poison, venesection (Olshausen, Wyder), diaphoresis in the form of wet warm compresses, according to Jaquet, and subcutaneous or rectal saline infusions have been recommended. *Prolonged chloroform narcosis* may directly influence the convulsions; some physicians have found chloroform-ether-narcosis, and especially the administration of morphia and chloral hydrate in large, or sufficient and repeated doses, to be successful, but the method is now rejected by many. Stroganow approves of the combination of all these measures. Nitroglycerine, thyroid extract (Nicholson, Baldowsky[3]), inhalation of oxygen, and other methods have been recommended.

Lumbar puncture is said to have had a good effect in a few cases (Helme[4]), whilst Henkel[5] in Olshausen's clinic, and Thies,[6] have found it to be of no avail.

LOCALISED MUSCULAR SPASMS

Facial Spasm

SPASMUS FACIALIS. CONVULSIVE TIC

The facial muscles are most frequently attacked by spasm. This is perhaps chiefly due to the intimate relations which exist between the mental processes and the movements of the facial muscles, and to the very rapid reflex action of these muscles.

Both these factors play an important part in the production of spasms.

[1] *Prag. med. Woch.*, 1901.
[2] *D. m. W.*, 1907.
[3] Abs. *Therap. d. Geg.*, 1904.
[4] *Brit. Med. Journ.*, 1904.
[5] *C. f. Gynäk.*, 1904.
[6] *C. f. Gynäk.*, 1906.

Painful affections of the conjunctiva and cornea, carious processes in the teeth, and any disease of the sensory trigeminus may give rise in this *reflex* manner to facial spasm. Facial spasm and tic douloureux are, therefore, not infrequently combined. When there is an existing predisposition, any local irritation may cause the development of a tic in the corresponding muscles—as Meige and Feindel have shown with regard to the localised and generalised tics (see chapter on general tic)—as the originally physiological reflex movements gradually develop into a habit spasm.

It is not quite certain whether affections of other parts of the nervous system may, in this way, give rise to facial spasm. This effect has been attributed to *diseases of the generative organs*, especially to uterine disorders, and in a few cases the results of treatment seem to indicate this connection. Gowers saw facial spasm develop during pregnancy and disappear after confinement. Bernhardt [1] has found similar connections between the disease and pregnancy.

In a great many cases facial spasm has a *psychogenic* origin, and has developed after some emotional shock or prolonged excitement. I have seen several persons suffer from convulsive tic after an earthquake.

Those affected are usually of a *neuropathic* disposition. I have seen convulsive tic develop from contraction of the orbicularis palpebrarum, which had either been voluntarily produced by a neuropathic patient in order to counteract a squint, or was of reflex origin.

Direct heredity is not common.

Irritation of the nerve trunk is the cause in a very small number of cases. Some observations show that compression of the nerve at the base of the brain (by a tumour or aneurism) has been the cause. It is not impossible, however, that even in these cases an overlooked compression of a sensory nerve may have *reflexly* produced the spasm. At least, I have seen spasm of the facial nerve on the same side occur in a case of intracranial tumour of the anterior cranial fossa, which had injured the first branch of the trigeminus.

A form of partial facial spasm has been described as watchmakers' occupation neurosis (T. Cohn [2]). On the other hand, predisposition to this affection is increased by overstrain of the facial muscles, the orbicularis palpebrarum in particular, which is caused by the patient's occupation.

Contracture resulting from *facial paralysis*, and accompanied by return of the power of movement in the facial muscles, is not a facial spasm in the strict sense of the word. But clonic twitching not infrequently occurs in the muscles previously paralysed, due possibly to fine changes in the nucleus which keep it in a condition of irritation (Hitzig, etc.). These twitchings may pass from one side to the other. On the other hand facial paralysis, due to ear disease, has been reported by aural surgeons to be occasionally preceded by a stage of clonic twitching. This has also been observed in traumatic irritation of the nerve trunk. Interesting observations of an apparently primary facial spasm, which were, however, from the first associated with paresis, are reported by Negro [3] and Thomas-Rieder.[4] Facial spasm in disease of the ear is some-

[1] See the article on facial spasm in *Nothnagels Handbuch*, second edition, 1904, which contains a large number of references to the literature.

[2] *N. C.*, 1897. [3] *Gas. degli Oped.*, 1906. [4] *R. n.*, 1907.

times of reflex origin and sometimes caused by direct injury of the peripheral nerves.

It is unusual for a spasm limited to the facial muscles to be due to an *organic disease of the cortex* in the region of the centre for this nerve; in these cases the symptom is usually one of cortical epilepsy beginning in the facial muscles, but subsequently extending to the arm and finally to the whole of one side of the body.

Facial spasm following *injury to the head* may be due to an organic disease, but it is so often also a symptom of traumatic neurosis that even when there has been an injury the symptoms may be partly of a functional nature.

We shall not here consider the conditions, such as epileptic or hysterical spasm, chorea, etc., of which facial spasm is one of the symptoms.

In the great majority of cases facial spasm has no pathological basis; it has been assumed that *minute* (? molecular) *changes* in the nucleus or cortical centre keep up the condition of irritation which is revealed by the spasmodic movements. A case published by Habel [1] seems to show that the spasm is not of cortical origin; in it the facial spasm persisted after a hemiplegia had developed on the same side.

Brissaud [2] has endeavoured to distinguish between *true* and *psychogenic* facial spasm. His theory has since been expanded by his pupils, Meige [3] in particular. When we have a muscular movement produced by emotion, a movement of expression, or a pathological habit which has become chronic, and is no longer a reflex movement produced by a peripheral irritation, we are dealing with a *convulsive tic*. On the other hand, *facial spasm* is a form of contraction which is independent of the mind, and is caused by dynamic or organic alterations in the reflex arc—the sensory trigeminus, the nucleus and trunk of the facial nerve. The indications for differentiation are contained in this definition. Tic has somewhat the character of gesticulation, shows intimate relations to the mental life, and is influenced by distraction of the attention; in spasm these relations are entirely absent, and the muscular contractions resemble those produced by a weaker or stronger electrical stimulation of the nerve or one of its branches. This character is specially marked when the spasm involves all the muscles innervated by the facial nerve of one side (the platysma included), and is clearly limited to them.

Meige draws attention to the manner in which the spasm takes its course. It usually, especially at first, comes on in attacks, separated by intermissions. The attack begins with a fibrillary tremor, or a vibration, *e.g.* in the orbicularis palpebrarum, which gradually increases in intensity and extent, until eventually a tonic spasm occurs, during which certain parts may still continue to quiver and twitch. Tic, on the other hand, always consists of the same gesticulatory movement of the muscles, which, of course, may vary in intensity.

Tic as a rule disappears during sleep, whilst spasm usually persists. Babinski [4] confirms the statements of Meige and Brissaud and points out other differential signs, viz., involvement of the aural muscles, distortion of the nose, and formation of dimples on the chin in facial spasm.

Symptoms.—Facial spasm, especially in Brissaud's sense, is usually limited to one side. It affects the whole facial region—although the

[1] *D. m. W.*, 1898.

[2] "Tics et spasmes clon. de la face." Leçon faite à la Salpêtrière, 1893; "Leçons," etc., 1895.

[3] *R. n.*, 1898 and 1903.

[4] *Nouv. Icon.*, xviii.; *R. n.*, 1905. See also Dupré, *R. n.*, 1903; Plavec, *W. m. Pr.*, 1904; Grolhouse, *Thèse de Paris*, 1904; Ingelrans, *Echo méd. du Nord*, 1905.

digastric and stylohyoid are usually, and the orbicularis oris often spared—or is limited to certain muscles or groups of muscles. The orbicularis palpebrarum is specially apt to be affected. The zygomaticus, the levator alæ nasi et labii superioris, and less often the orbicularis oris and the chin muscles, may each be independently involved. A spasm affecting only the muscles of the chin (geniospasm) has been described by Massaro[1] as a hereditary disease. These twitchings, which are persistently limited to a small muscular area, should in most cases be regarded as tic. I have occasionally seen a convulsive tic limited to the muscles of the nose, and still more rarely a tic confined to the frontal muscles. A snuffing-tic has been described by Meige and Feindel,[2] and by Bechterew, and I have also seen it. In a case described by Stembosch, in which the twitchings were limited to the occipito-frontalis muscle, hysteria was the cause. The twitching is much more rarely limited to the external muscles of the ear (Romberg). Bernhardt observed spasms in the occipitalis muscle, and the retrahens auriculæ. Diffuse spasm extends to the platysma, and not infrequently to the ear muscles. I have lately seen a spasm limited to the platysma, consisting of clonic twitching occurring simultaneously or alternately in both platysmata, in a boy who could voluntarily contract the platysma independently of other muscles.

The spasm is usually *clonic*, but clonic twitching may be associated or alternate with *tonic* contraction. In tonic contraction the palpebral apertures are contracted until the eyelids are closed, whilst the forehead is thrown into furrows, and the nose and mouth are drawn towards the side of the spasm. The face is greatly wrinkled. The clonic twitchings are often so slight that they can only be detected by close observation, but at other times they are so marked as to produce a constant play of lively grimaces. Short lightning-like quivers are rapidly repeated or pass only once over the muscle. As a rule there are *paroxysms*, in which the twitching follows in quick succession, whilst in the intervals the muscles are entirely at rest, or show merely slight, infrequent contractions.

In complete mental and physical rest the twitching is usually least marked, and it is exaggerated by *mental excitement*, by *exertion*, in particular by *eating* and *speaking*, and by contact with cold air. The fact that convulsive tic diminishes when the patient's attention is distracted, that it may be temporarily inhibited by suggestion, etc., indicates its dependence upon *mental* processes. I have seen the tic in a celebrated pianist keep time exactly to his playing. Facial spasm is subject to *spontaneous* remissions, intermissions, and exacerbations, but it may also be aggravated by great excitement, over-exertion, etc. The spasm may, as a rule, be easily elicited by sudden examination of the patient's eye, when the closing of the eyelid usually develops into spasm, by tapping the face, or rapidly passing the fingers over the skin of the face.

In some cases—especially of *blepharospasm*, *i.e.* tonic spasm limited to the muscles which close the eye—*pressure-points* may be found in the trigeminal region (Graefe), at the point of emergence of the supraorbital, etc. Slight pressure upon these points has an *inhibiting action upon the spasm*, the muscles which were tonically contracted becoming suddenly relaxed. As a rule, however, this is merely the effect of suggestion, and is a symptom found only in tic. Such pressure-points are occasionally found at distant sites, *e.g.* on the vertebræ. Spasm of the eyelid is usually

[1] *Il Pisani*, 1894 (*R. n.*, 1894).

[2] "Les Tics et leur Traitement," Paris, 1902.

of a tonic nature (blepharospasm), but the clonic form (blepharoclonus, nictitatio) is also not uncommon.

The former may be so persistent that the eyes remain closed for weeks and months. The fact observed by Graefe that transient blindness may occur in children after longstanding blepharospasm, is a very interesting one; the nature of this blindness is not yet explained, in spite of numerous investigations (Silex,[1] Samelson,[2] Baas, and others).

I have seen a patient with bilateral blepharospasm, in whom the spasm stopped for a moment when the mouth was opened, possibly on account of the occurrence of an associated movement in the levator palpebræ superioris (see p. 86).

Facial spasm is accompanied sometimes by slight, sometimes by very severe subjective disorders. This depends chiefly upon the intensity of the spasm and upon whether the orbicularis palpebrarum is involved or not. It is surprising how very well some patients can bear this affection; thus, I have repeatedly noted the clonic form in working people who were quite unaware of its existence. On the other hand true facial spasm may be associated with, or provoked by pain in the trigeminal region. The voluntary movements of the facial muscles are not as a rule affected, but the spasm may occur during these movements and disturb their course, and voluntary movements may also provoke or increase the spasm.

Symptoms pointing to involvement of the *stapedius muscle, e.g.* noises in the ears, which in exceptional cases can be heard by other people, are very uncommon.

The *soft palate* is hardly ever affected in pure facial spasm. In a few cases in which twitching of the uvula was observed, *e.g.* by Leube,[3] Schütz,[4] and Schüssler,[5] some complication may have been present.

In cases of spasm of the soft palate (*spasmus palatinus*) under my own observation (*N. C.*, 1889), an organic disease was usually present—a cerebellar tumour in one case and previous epidemic cerebro-spinal meningitis in another. The vocal cords or the internal laryngeal muscles contracted synchronously with the soft palate. Sinnhuber (*B. k. W.*, 1904) has described a similar condition and referred to a case of Spencer's (*Lancet*, 1886). Siemerling and I (*Charité-Annalen*, xii.) have also seen isolated twitching of the soft palate in aneurism of the vertebral artery. Meyer and also Klien (*D. m. M.*, 1904) describe similar cases. Twitching limited to the soft palate and usually associated with crackling noises has also been observed (Rosenthal, Williams, Peyser [6] and Avellis, Donath, Oppenheim, Roemheld,[7] Goldflam-Meyerson,[8] Valentin,[9] Lachmund,[10] etc.). In a case of this kind described by Bernhardt (*D. m. W.*, 1898), the muscles of the larynx and tongue were involved in the twitching. The noise which is perceived in this spasm of the palate is due either to contractions of the tensor tympani or of the tensor palati which separate the two lips of the tubal orifice from each other. This form of spasm may also be included under the heading of trigeminal spasm.

Vasomotor, trophic, and secretory disturbances are hardly ever present.

In a few rare cases (Bernhardt,[11] Newmark,[12] Vitek,[13] Spiller, Oppenheim, H. Frenkel [14]), a *myokymia* (*q.v.*) limited to the muscles supplied by the facial has been observed, but Meige will not admit that this represents a special form of spasm.

[1] *Zehenders klin. Monatsh.*, 1888.
[2] *Ibid.*, and *C. f. prakt. Augen*, 1888.
[3] *Bayer. ärztl. Intellig.*, 1878.
[4] *Prag. med. Wchnschr.*, 1882.
[5] *B. k. W.*, 1879.
[6] *B. k. W.*, 1895.
[7] *M. m. W.*, 1903.
[8] *W. m. Pr.*, 1895.
[9] *Z. f. Ohr.*, Bd. xlvi.
[10] *M. f. P.*, xxi.
[11] *N. C.*, 1902.
[12] *N. C.*, 1903.
[13] *N. C.*, 1904.
[14] *R. n.*, 1903.

Direct Treatment.—Drugs on the whole have little effect, but arsenic and bromides may be helpful. They are most likely to be so in convulsive tic. Quinine, atropin, cannabis indica, tincture of gelsemium, etc., are hardly ever of use. Hyoscin may produce temporary improvement. In a few cases dropping of cocaine into the nose, conjunctiva, or ear (Rampoldi) has had a soothing effect. Bloch,[1] in one case, found subcutaneous injection of antipyrin into the affected side of the face to be successful, but it also caused paralysis of the muscles.

Within the last few years the method specially recommended by Schlösser,[2] of the injection of alcohol (70 to 80 per cent.) into the nerve sheath, has also been used for facial spasm. The paralysis which it at first produces passes rapidly away. Successful cases have been reported by Schlösser, Brissaud-Sicard-Tanon,[3] Lévy-Baudouin,[4] Abadie and Dupuy-Dutemps,[5] and by Patrick.[6] Schlösser recommends that the syringe be pushed along the stylomastoid process as far as the base of the skull: a few drops are at first injected, and the injections are then continued with short interruptions until distinct paralysis is produced. The recovery which took place in the majority of his cases lasted for three to seven months, when a relapse occurred which necessitated repetition of the treatment.

Diaphoretic measures are suitable in recent cases, and *counter-irritants* may be used in the form of a cantharides plaster behind the ear. *Electrotherapy*, especially galvanism, is certainly successful, though only in some cases. Various methods of treatment may be tried, *e.g.* an anode of about 10 sq. cm. may be placed on the nerve trunk, the cathode on the nape of the neck or some indifferent spot, a weak current of 2 to 3 milliamperes being slowly turned on and off; the anode may be applied to the occiput, the cathode to some distant part, or both electrodes to the mastoid process; the anode to the various branches of the pes anserini major, etc. When pressure-points can be discovered, it is advisable to apply the anode to them, in order to diminish their excitability. The application of the anode to the cortical centre of the facial, therefore to the opposite parietal bone, has even been recommended. The faradic current gradually increased, the static breeze, and the Arsonval current have also been advised.

I have found gymnastic exercises successful in a few cases, especially the *inhibition treatment* which I have recommended (see chapter on general tic).

It has been thought advisable to *operate* in some obstinate cases, and especially to resect the *supraorbital*, when this nerve has been tender to pressure and when pressure upon it inhibited the spasm. In a few cases this has been followed by permanent recovery. Simple section of the nerve seems to be of no avail. In other cases the facial nerve has been *stretched*. The result was paralysis instead of spasm, and as the paralysis disappeared, usually within a few months, the spasm again made its appearance (Bernhardt[7]). In one case only is the recovery said to be complete two years later (Southam). This method may be tried in particularly persistent cases, in which it would be a relief to the patient to be

[1] *A. f. P.*, Bd. xxxviii.; "Bericht. d. Ges. f. P."

[2] "Bericht. d. 31. Versamml. d. ophth. Ges. Heidelberg"; abs. *B. k. W.*, 1904; also *B. k. W.*, 1906.

[3] *R. n.*, 1907. [4] *R. n.*, 1906. [5] *R. n.*, 1906. [6] *Journ. of Amer. Med. Assoc.*, 1907.

[7] *A. f. P.*, xv., and Nothnagel's "Handbuch," *loc. cit.*

free even for a few months from the troublesome twitching, but the patient should be warned that the spasm will be replaced by paralysis. The alcohol infiltration of the nerve, mentioned above, may then be preferred to nerve-stretching. The case cited above shows, however, that even in severe cases there is a possibility of spontaneous recovery.

Kennedy[1] sections the facial nerve and grafts it on to the spinal accessory, which is cut for the purpose. It cannot yet be determined whether this method deserves to be imitated, but it seems to me unlikely.

Blepharospasm calls for treatment of the ocular trouble. The symptoms can often be relieved by dropping *cocaine* into the eye. The sudden immersion of the face in cold water may for a time soothe the spasm. Wolfberg found binding up the unaffected eye to be of use in unilateral blepharospasm. As this twitching is often of a hysterical nature, it may yield to any method of suggestion, as in a case under my care, in which the trouble had persisted for over twenty years. What has been said with regard to facial spasm applies in general also to blepharospasm.

Spasm of the Muscles of Mastication

Spasm limited to the motor trigeminal nerve is not common. We must distinguish between the tonic and the clonic forms. In tonic spasm the jaws are usually tightly clenched, the masseters and temporal muscles stand out prominently and feel as hard as a board. The patient can either not separate his teeth at all, or does so only with great difficulty, and passive attempts to separate the jaws are met with strong resistance. If the spasm persists for any length of time, the nutrition naturally suffers. Lateral displacement of the lower jaw is a rare occurrence; in unilateral spasm of the pterygoids, the lower jaw is displaced towards the opposite side. *Trismus* is a symptom of *tetanus* or *meningitis*; it occurs less frequently in *tetany*, or as a transient symptom in the tonic stage of an *epileptic* attack. Tonic spasm of the muscles of mastication has repeatedly been observed in diseases of the pons, in commencing *acute bulbar paralysis*, and in tumours. It may undoubtedly be caused by irritation of the cortical centres (Lépine, *Rev. de Méd.*, 1882).

It is very seldom an *isolated* symptom. It has usually a *reflex* origin, and is due to inflammatory conditions of the temporo-maxillary joint or its neighbourhood, or of the mucous membranes of the mouth, to a carious wisdom-tooth, or to difficulty in its cutting through the gum. Trismus of myogenic or myositic origin has also been described. In one case which I saw there had been a septic wound; the general condition was good, and the trismus seemed to be the only symptom of tetanus. Eiselsberg (*D. m. W.*, 1898) has also seen this in one case, but he regards the masseter spasm as being due to hysteria.

Trismus is not an unusual symptom in *hysteria*. In a case reported by Bidlot-Francotte (*Journ. de Neurol.*, 1897) hysterical trismus lasted for nine months. In one of Chatin's (*R. n.*, 1900) the psychogenic origin was indicated by the fact that the patient could only stop the twitching by placing his finger between his teeth, and could not suppress the muscular tension in any other way (mental trismus). Raymond and Janet report a similar case. Leube (*A. f. kl. M.*, vi.) observed unilateral spasm of the pterygoids in a girl who suffered from hysteria and chorea. Tordeus (*Journ. de clin. et thér. inf.*, 1897), Féré, and others have described tonic spasm of this kind. It may be caused in nervous people by some violent emotion (terror). A few cases point to a rheumatic origin. Kocher would distinguish trismus as an idiopathic spastic neurosis from hysteria.

Clonic spasm produces *rhythmic movements of the lower jaw* in the vertical, rarely in the horizontal direction. These are sometimes so strong that they make the teeth chatter as in a rigor. The muscles of mastication are usually involved in general convulsions (hysteria, epilepsy, etc.), and the tremor of paralysis agitans is sometimes localised in these muscles. Clonus confined to the jaw muscles is a *rare* symptom. It has been repeatedly observed in hysteria, usually as a *temporary* symptom. But I have treated a young girl who for many years had been

[1] *Philos. Trans. Roy. Soc. of London*, 1901.

greatly distressed by clonic twitching of the muscles of mastication. This became so markedly increased in speaking that she spoke like a person in a rigor, and had difficulty in taking food. In the forms of disease in which the "*feed-reflex*" which I have described occurs, especially in infantile pseudo-bulbar paralysis, there is spasm of the masseter muscles which appears to be spontaneous, but is mostly reflex, and is due chiefly to an accumulation of saliva.

A slight form of this spasm, viz., grinding the teeth, is not uncommon in nervous children, and even in adults during sleep. Spasm in the muscles which depress the inferior maxilla, giving rise to a kind of trismus, has been observed by Oppenheim, Placzek (*B. k. W.*, 1898), and others after a paroxysm of trigeminal neuralgia. Spasm of the masseter and facial muscles, apparently the reflex result of a disease of the ear, has been observed by Wagner (*D. m. W.*, 1907).

Spasmodic conditions may also occur in the tensor tympani muscle, which give rise to noises in the ear (compare the foregoing chapter). The movement of the tympanic membrane may be detected by otoscopic examination (Schwartze and Burnett).

The *prognosis* of spasm of the muscles of mastication is good, if an organic disease can be excluded. It usually disappears within a few weeks or months, although stubborn cases do occur.

Treatment should have regard to the reflex origin of the condition, and should be directed to any inflammatory process or other conditions of irritation in the region of the trigeminal nerve. If the irritation arises from a decayed tooth or an ulceration within the mouth, these should be suitably dealt with. If the trismus prevents access to the mouth, an anæsthetic should be given. Trismus of long duration calls for artificial feeding (by a nasal tube). In recent cases in which a rheumatic influence may be at work, diaphoretics or counter-irritants (cantharides to the forehead or on the mastoid process, or a button cautery to the nape of the neck) are advisable. The various sedatives and nerve tonics may also be prescribed. Galvanism may have a curative effect.

I have occasionally seen trismus relieved by pressure on the ovaries. Psychotherapy and inhibition exercises may also be employed.

Spasm of the Hypoglossal Region. Spasm of the Tongue. Glossal Spasm

The general convulsions of epilepsy and hysteria may involve the muscles of the tongue, which usually also participate in the spasms of chorea, but are very seldom independently affected. The spasm of the tongue may be *tonic* or *clonic* in nature, and a mixed form has also been observed. In tonic spasm the tongue is hard and firm, is decreased in diameter and is pressed against the palate or teeth. Speech and deglutition are impaired during the spasm, but respiration is seldom affected. The speech trouble, which is due to sudden and very transient tonic contraction of the muscles of the tongue, is known as *aphthongia* (Fleury [1]). Other muscles, *e.g.* those of the face and eyes, etc., may be involved (Steinert [2]). The most common cause is emotion, less often some reflex irritation, such as an affection of the throat. This form of spasm, as Gutzmann [3] rightly states, is closely allied to stuttering, or is merely a variety of it. Lange's [4] case, in which a tonic spasm of the tongue, which kept the tongue almost constantly protruded beyond the teeth, disappeared in eating and speaking, is a very exceptional one. The clonic twitching moves the tongue rapidly or slowly in and out of the mouth, or it may remain inside the mouth and be moved from side to side and round its longitudinal axis. Both sides of the tongue are as a rule equally involved, but a hemispasm which causes the tongue to be obliquely protruded may occur, *e.g.* in hysteria. A combination of unilateral glosso-spasm with fibrillary tremors of the muscles has been described. The spasm may extend to other muscles, especially the inferior facial.

The spasm is seldom continuous; it usually comes on in paroxysms which occur every few weeks or days, 20 to 30 times a day—seldom during the night—or even every 5 to 10 minutes. The attack may only last for a few seconds or minutes, or may continue for hours. It does not always cease during sleep. The spasm of the tongue is sometimes caused by peripheral irritation, *e.g.* inflammation of the mouth, carious teeth (Mitchel), extraction of a tooth, neuralgia of the tongue, etc. In one case a foreign body embedded in the occipital nerve was the cause.

[1] *Gaz. hebd.*, 1865.
[2] *M. m. W.*, 1902.
[3] "Monatsschr. f. d. ges. Sprachheilk.," 1898.
[4] *Langenbecks Arch.*, Bd. xlvi.

As a rule such causes cannot be discovered. The *neuropathic* disposition is a very important factor. Thus the spasm frequently occurs in *hysteria, psychasthenia, and hypochondria,* and in persons who have been or are epileptic. This form more often assumes the character of a tic, in Brissaud's sense, than of a spasm. The neuropathic diathesis is indicated by the combination, which I have several times found, of spasm of the tongue and *mental changes.* In one hypochondriacal, weak-minded patient the twitching movements involved simultaneously the muscles of the tongue, jaws, and lips; in another case the muscles of the tongue and of the jaws were alternately affected, and in a third, in which the trouble followed the extraction of a tooth, the muscles on the floor of the mouth were also involved, but the hypochondriacal ideas which were present indicated that the condition was of mental origin. Sepilli (*Riv. sper.*, 1886) saw the spasm accompany a puerperal psychosis. Strümpell [1] mentions an occupation spasm of the tongue in a glass blower.

Paræsthesiæ and pain in the tongue sometimes precede the twitching. The spasm is very often caused by emotion. It may persist for months or years, but in the greater number of the published cases recovery has ultimately taken place.

The *prognosis* is not grave. The spasms may persist for years, but in most cases recovery ultimately takes place.

Treatment.—Treatment of the general health is of special importance; the system should be strengthened by tonics, dieting, hydrotherapy, etc. Sedative drugs should also be prescribed. Personali recommends antipyrin in increasing doses. If there is any reflex cause, it should if possible be removed. Galvanism, applied as for facial spasm (anode on the hypoglossal nerve) should be tried in every case. One patient recovered after a sea-voyage, although every other remedy had failed. Psychotherapy should never be neglected in this form of spasm.

Operative measures are hardly ever necessary, but in Lange's case the hypoglossal nerve was stretched, then resected, and finally the geniohypoglossus muscles were divided, and this operation proved successful.

Spasm in the Muscles supplied by the Glossopharyngeal Nerve. Deglutition Spasm. Pharyngismus

These spasms hardly ever occur independently. They develop either in hysteria, hypochondria, and psychasthenia, or form a symptom of rabies and hydrophobic tetanus. I have recently seen a patient in whom clonic spasm of the muscles of deglutition, occurring chiefly during eating, especially when in company, had been for years a very distressing symptom. He also suffered from hay-fever, an affection which in my experience develops only in neuropathic persons. Spasm of the muscles of deglutition may occur in the course of tabes in the form of pharyngeal crises. I have once seen a similar phenomenon in bulbar gliosis. Local diseases of the pharynx and œsophagus, *e.g.* carcinoma, may also produce spastic conditions in these muscles.

Spasms in the Muscles of the Neck

For literature see the text-books and monographs of Romberg, Erb, Gowers, Tillaux, Brissaud, Henoch, Finkelstein; also Fournier, "Le Tic rotatoire," *Thèse de Strassbourg,* 1870; Isidor, *Thèse de Paris,* 1895; Bompaire, *Thèse de Paris,* 1897; Feindel, *Nouv. Icon.,* 1897; Quervain, *Semaine méd.,* 1896; Redard, "Le Torticolis et son Traitement," Paris, 1898; Raudnitz, *Jahrb. f. Kind.,* Bd. xlv.; Cruchet, *Thèse de Paris,* 1901-1902; Kalmus, "Beitr. zur kl. Chir.," xxvi.; Meige-Feindel, "Les Tics et leur Traitement," Paris, 1902; Jacquet, *Thèse de Paris,* 1903; Stamm, *A. f. Kind.,* xxxii.; Ziehen, *Z. f. prakt. Ärzte,* 1903; Still, *Lancet,* 1906; Rietschel, *Charité-Annalen,* xxx.; Cruchet, "Traité des Torticolis spasmod.," Paris, 1907; Steyerthal-Solger, *A. f. P.,* Bd. xxxviii.; Kocher, "Chirurg. Operationslehre," 5th ed., 1907; Bernhard, Nothnagel's "Handbuch," xii., 2nd ed., 1904; Meige, *Nouv. Icon.,* 1907.

The spasm which affects the muscles of the throat and neck is a very serious form on account of its intractability and the injurious effect which it has upon the general health.

It may be unilateral or bilateral, and may be limited to individual muscles of the neck, to several muscles of one side, or may involve a

[1] *Spez. Path. u. Therap.*, iii., 1900.

number of muscles of both sides. The muscles innervated by the *spinal accessory* are specially liable to be affected, but one is hardly justified in differentiating this form as an " accessory spasm," and in discussing it separately, as it differs from the other forms neither in its symptoms, prognosis, nor treatment. Thus the *sternomastoid* may be affected alone or in conjunction with the *trapezius*, or the sternomastoid and *splenius* of the same or opposite sides of the body, the splenius and trapezius, or the *scaleni* and *deep cervical* muscles may be simultaneously involved; in short, every imaginable combination may occur. The *platysma myoides* and the *omohyoid* occasionally participate in the twitching. These spasms do not as a rule show any tendency to become limited to the area supplied by a certain nerve, but are apt to begin in one muscle and to spread to others in the course of the disease.

The trouble chiefly affects individuals of a *neuropathic* and *psychopathic* constitution.[1] Direct heredity or a family occurrence is, however, very rare. An interesting case of this kind has recently been published by Steyerthal and Solger. The relation of the spasm to the neuropathic diathesis is indicated by its frequent combination with other *neuroses*, and in particular with *psychoses*. The spasm shows frequent associations or alternation with mental disturbances, and the so-called stigmata of degeneration are commonly found in those affected. This is particularly the case as regards true tic of the cervical muscles (*q.v.*), whilst spasm of these muscles may occur in healthy persons with no neuropathic tendencies.

Rheumatism of the cervical muscles, *rheumatic torticollis*, *i.e.* wry-neck produced by contracture of the rheumatic or myositic sternomastoid (known also as rheumatic caput obstipum), gives rise to a stiff carriage of the head, but is not a spasm in the strict sense of the word. Wry-neck, whether congenital or acquired during parturition, need not be discussed here.

The condition is seldom due to *trauma*, or to a *reflex* cause, for the muscular contraction which develops in diseases of the cervical vertebræ (especially caries) is an *accidental* condition and has nothing in common with spasm of the cervical muscles properly so called. Irritative conditions in the region innervated by the trigeminus and the occipital nerves (Mills, Ziehen) have, however, been regarded as a cause. Indeed, any peripheral irritation, even a tight collar, may in *predisposed* individuals be the first exciting cause of the spasm. Ziehen speaks of " *desequilibrating* " causes (to which Tillaux had previously referred); among these, he includes cases in which the " symmetrical proportion of the excitations " of the central nervous system is destroyed by some unilateral disease or by the excessive demand made upon one of the special sense organs, the innervation which has previously been symmetrical being thus made

[1] In one case under my observation the grandparents of the patient were related to each other; the grandfather had diabetes, the grandmother was insane, the mother nervous, some of the children epileptic, the others mentally defective. The patient suffered in her youth from *general tic*, which disappeared for some years, but after her marriage a spasm of the spinal accessory developed. In another case I saw this symptom (combined with facial spasm) appear in a man of 70 who denied any hereditary predisposition, but I afterwards discovered that he had a general neurofibromatosis, and that an uncle suffered from Friedreich's disease. In the case of a lady who for 20 years had been afflicted with this spasm in the severest form I had ever met, I found a peculiar malformation which I had once before seen in a slighter degree, viz., the insertion of the sternomastoid to the sternum was continued in long, tendon-like processes, which crossed above the manubrium sterni and could be followed into the mammary region.

unequal. Amongst such cases are, *e.g.*, the rare ones in which paralysis of the ocular muscles first causes secondary rotation of the head, followed by chronic torticollis (Nieden,[1] Dallwig [2]), or which are due to a professional rotation of the head to one side (Duchenne, Grasset, Baylac [3]). Ziehen suggests that vasomotor disturbances, in particular asymmetry in the circulation of the blood in the nuclei of the corresponding nerves, may bring on the spasm.

Torticollis in diseases of the ear, in which there is usually direct extension of the inflammatory or suppurative process to the muscles, or less commonly reflex muscular contraction, is not an independent disease. Curschmann [4] has recently described cases of this kind in which spastic torticollis could be traced to disease of the labyrinth. Observations of this kind had already been reported by Okouneff.[5]

Organic diseases of the brain may give rise to spasm in the cervical muscles, but this is uncommon. I have seen such spasms once in tumour of the cerebellum which compressed the medulla oblongata and the nerves arising from it, and several times in cysticercus cerebri.

Primary atrophy of the sternomastoid of one side is said to produce spasm in the muscle of the other side (Féré, *Rev. de Méd.*, 1894). Lengemann describes clonic-tonic spasms in the right sternomastoid in a patient who had a cartilaginous tumour in the left sternomastoid. This was therefore a case of the desequilibration form of Ziehen.

Chronic poisoning also seems to play a part in the etiology. I have seen the spasms follow alcoholism and chronic metallic poisoning. Guibert [6] regards toxicopathic heredity as a cause. One intermittent case was due to *malaria* and was cured by quinine. In another case under my personal observation the spasm was directly due to *influenza*. It has been seen to follow typhoid and pneumonia. The combination with diabetes, which I have found in one case, was explained by the neuropathic diathesis. Chill, trauma, and over-exertion are often reported as causes, and overstrain in the muscles of the throat and neck resulting from errors of refraction, astigmatism (Walton [7]), working in badly lighted rooms (Raudnitz, Rietschel), or professional exhaustion of the cervical muscles are very apt to cause the spasms. Thus, in several cases they came on during reading or writing (Duchenne, Oppenheim, Näcke [8]). Rickets is also thought to favour the onset of the infantile form.

There are other cases for which no cause can be discovered. Some have been attributed to "a bad habit," but I am of opinion that ineradicable habits of this kind only develop in neuropathic individuals. Examples of this kind, in which an originally purposeful reflex movement of defence or gesture developed into torticollis, have been reported by Raymond-Janet, Meige-Feindel, Sgobbo,[9] and others.

We have no hesitation in saying that the primary cause in typical cases is the *neuropathic* or *psychopathic diathesis*, and that, given this constitution, a number of factors, *e.g.* mental excitement, trauma, over-strain of the cervical and nuchal muscles, may bring on the spasm. In this

[1] *C. f. Aug.*, 1892.
[2] "Inaug.-Diss.," Marburg, 1897, and *A. f. Aug.*, Bd. xxxvi.
[3] *Arch. méd de Toulouse*, 1903; *R. n.*, 1904.
[4] *Z. f. N.*, xxxiii.
[5] *Arch. internat. de Laryng.*, 1904.
[6] *Rev. de Méd.*, 1892.
[7] *Journ. Nerv. and Ment. Dis.*, 1897.
[8] *N. C.*, 1906.
[9] "Il Manicomio mod.," 1898. See also Gaussel, *Nouv. Icon.*, xvii.

opinion we are practically at one with Brissaud, who regards the condition as a psychogenic one.

After the muscles of the face, those of the neck are most implicated in the movements of expression. Moreover, they are in a constant state of activity, as they are required not only to keep the head erect, but to bring about its almost ceaseless play of movement. It is, therefore, easy to understand why these muscles are specially liable to be affected in the transformation of mental processes into motor acts. Nor can there be any doubt that conditions corresponding to pathologically fixed ideas and memory images (refer to chapter on imperative ideas) come into existence in the motor-kinæsthetic system, that there is a close relationship between these, and that localised muscular spasm frequently owes its origin to the fact that some emotional process, instead of becoming fixed in the mind as an imperative recollection, immediately invades the motor sphere and discharges itself in the form of a motor action. It is still uncertain whether relations of contiguity—the entirely hypothetical localisation of the centres for the mental processes and those for the muscles of the neck in the frontal lobe—may also play a part.

We should also mention the occurrence of cases in which there is no evidence of a psychopathic heredity or tendency, and to which Brissaud's interpretation does not apply, as his pupils, Meige and Feindel, themselves admit. Tic may also be associated with a true spasm of the neck muscles (see preceding chapter). The latter group probably includes most of the cases of infantile spasm of the cervical muscles, the spasmus nutans of early childhood (*q.v.*), which seem to be chiefly due to dentition and other reflex causes.

Symptomatology.—There is a tonic, a clonic, and a mixed form of these spasms. As a rule *clonic* twitching is associated with transient tonic contraction of the muscles. Prolonged tonic spasm of the sterno-mastoid, with contracture and consequent oblique position of the head, is rarely primary, but is more probably the result of rheumatism, or due to disease of the cervical vertebræ and their vicinity, to traumatic myositis terminating in fibrous atrophy, or finally to a *congenital* shortening of the muscles. Tonic spasms may, however, take the form of an attitudinal tic (Meige-Feindel).

The symptoms are dependent upon the *localisation*, form, and *intensity* of the spasms. If one sternomastoid only is affected, the face is turned towards the other side, the ear approached to the clavicle, and the chin raised; in spasm of the left sternomastoid, therefore, the face is turned upwards and to the right, whilst the left ear approaches the internal end of the left clavicle. If the spasm is slight, there are only feeble movements of rotation. The trapezius of the same side is often involved along with the sternomastoid; as a rule, only its *upper portion* is affected, the head being thus rotated towards the opposite side, and at the same time thrown backwards, the occiput being approached to the scapula. The spasm may occur simultaneously or alternately in the two muscles. If both trapezii are involved, the head is simply dragged or thrown backwards. Under these conditions the frontales are sometimes involved in the spasm (Gowers). At the height of the attack the spasm may extend to other muscles of the trunk and extremities. Indeed, there is a special type characterised by this mode of extension and by a gradual progression of the disease.

I have seen a very unusual form and extension of the spasm in an otherwise healthy man, who had suffered from the same condition some years previously. The middle part of the trapezius was mainly affected by a tremor which attacked the various bundles of the muscle in rapid

succession, and simulated a kind of division of the muscle (myoschisis, Fig. 414). The spasm differed from fibrillary tremor in that it involved, not muscle fibrils, but large bundles or dentations, and that it consisted, not of one continuous wave, but of a short contraction. Another peculiar point was, that raising the arm or stretching it forward was always followed by a rapid contraction, sometimes bilateral, which elevated the shoulder and notably the scapula.

The spasm is not infrequently limited to one *splenius* muscle; the head is drawn backwards and rotated towards the side of the contracted muscle.

Tonic spasm in the rhomboid and levator anguli scapulæ may give rise to " high position of the shoulder blade " (Eulenburg).

The spasm may consist in simple nodding movements—the *nodding*

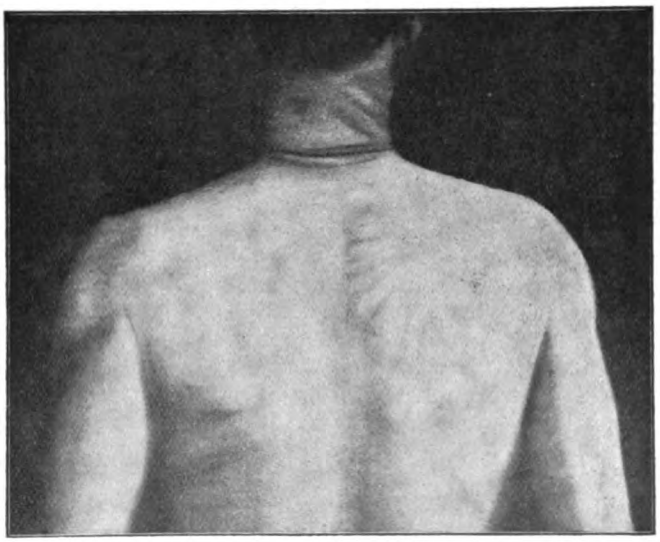


FIG. 414.—(Oppenheim.) Spasm of the right trapezius, most marked in its middle portion.

or *salaaming spasm* which is specially apt to occur in children at the time of dentition. It may be caused by clonic contractions in the *deep cervical muscles* (*recti*, *longus colli*, etc.), but the sternomastoids may also be involved. Rotatory movements may also appear. This *spasmus nutans* (nodding spasm), first described by Barton, Bennet, and Newnham, is usually combined with nystagmus, which specially occurs when the child's head is fixed. In two children under my care the nystagmus was limited to one eye—a symptom which it is difficult to explain. *Strabismus* and *blepharospasm* may also be present. The disease, which may have a familial form, attacks children during the first years of life. I have seen two cases of this kind in which the nodding spasm was only present during the night when the child was asleep. One of these patients was a girl of eleven, who came of a very neuropathic family (the father had six fingers and toes, and numerous members of the mother's family had spasm of the cervical muscles, particularly in this nocturnal form). The condition had developed in her earliest childhood, at which time there were also

movements resembling those of a nodding mandarin during the day, whilst later only the nocturnal or sleep torticollis had persisted.

This reference, already made in the third edition of this text-book, remained unnoticed until Zappert (*Jahrb. f. Kind*, Bd. xlii., and *W. kl. R.*, 1905) drew attention to the condition of "jactatio capitis nocturna" or "tic de sommeil," which I had first described. The symptom was subsequently studied by Cruchet (*Gaz. hebd. de Bordeaux*, 1904; *Presse méd.* 1905), Lange, Swoboda (*W. kl. R.*, 1905), Dercux (abs. *R. n.*, 1907), and Segre (*Arch. di Psich.*, Bd. xxviii.), most of whom have ignored my publications. Among my later cases the following is of special interest: A boy of 13 from the Argentine was brought to see me; since he was a year old he had every night for hours carried on rocking movements of the head and upper part of the body, usually in the sitting position. The condition could not be cured. He was otherwise healthy, and learned his lessons well. His mother had been married at the age of 13, and at 19, in the pregnancy preceding his birth, had become insane from hearing of the suicide of her father. In another of my patients, in whom the nocturnal rotation of the head was always directed towards the left, the spasm was sometimes so severe that vomiting occurred.

Simple *rotation spasm* (tic rotatoire, Schüttel-Tic), which sometimes occurs, is due to spasm of the *inferior obliquus* muscle. It is obvious that the condition may vary in numerous ways.

The involvement of the vocal cords and soft palate in spasm of the spinal accessory, observed by Gerhardt,[1] is very uncommon.

The intensity of the spasm varies greatly. It is sometimes so slight that it is uncertain which muscle is affected; in other cases it is so severe that the head is forcibly thrown backwards and forwards, and eating, speaking, and sleeping are all rendered difficult. It may also lead to compression of the supra-clavicular vessels and nerves (Romberg). The muscles chiefly affected may become *hypertrophied*. The spasms are *increased* by *emotion*, self-observation, and usually by any attempt to control them. Physical, and especially mental rest, and distraction of the attention, have an alleviating effect. In some cases the spasm ceases entirely when the patient lies down, returning as soon as he stands and walks. Ziehen calls this a co-intentional torticollis, but this term applies only to "mental torticollis." Brissaud points out that some patients can arrest the spasm by placing a finger on the chin. "Efficacious antagonistic movements" are found in most forms of tic (Meige-Feindel).

The spasm is in some cases continuous, although with remissions and exacerbations; in others it takes the form of attacks, separated by intervals of complete rest. Ten to thirty contractions may occur to the minute. *Sleep* does not always bring cessation, even in the cases which do not belong to the special nocturnal form (see above).

The patient does not as a rule complain of pain, but a dragging pain in the neck may precede or accompany the spasm. There are no symptoms of paralysis or changes in the sensibility or the functions of the special sense organs.

Mental disturbances, as we have already said, are frequently associated with these spasms. Romberg has already quoted a case of Brodie's, in which a mental disorder was to a certain extent an equivalent of the spasm of the accessory, which ceased so long as the mental disorder lasted. I have observed a similar *alternation of spasm in the muscles* of the neck and *hallucinatory confusion*. In another case under my care the spasms developed after delirium (probably of an alcoholic nature). Gowers has described a case in which *melancholia* was present ten years

[1] *M. m. W.*, 1894.

before the onset of the spasm. One of my patients, who at first denied any mental disturbance, subsequently reminded me that I had treated him a number of years previously for a marked condition of anxiety, and that two of his brothers were insane. In another, recovery from spasm of the spinal accessory, which had lasted from childhood, was followed by the development of fixed ideas. In a case described by Manasse, surgical treatment was followed by transient psychosis.

The combination with writers' cramp has been mentioned by Duchenne, Quervain, Destarac, Bonnus,[1] and Pitres.[2]

Differential Diagnosis.—Rheumatic torticollis is distinguished from true spasm of the cervical muscles by severe pain and marked tenderness of the muscles to pressure and on passive movements. The torticollis which is present from birth is characterised by its congenital development, the secondary changes in the cervical vertebræ, the shortening of the muscle, and the mechanical fixation of the head.

The review by Zesas, *C. f. Gr.*, 1905, gives a good summary of our knowledge of congenital "muscular wry-neck."

Persistent wry-neck always suggests a *local disease*, in particular one of the cervical vertebræ. It should also be borne in mind that an organic brain disease (especially a tumour or cysticercus) may be the cause. These conditions may be excluded by careful examination.

General tic (*maladie des tics convulsifs*) and *myoclonia* may commence with spasm of the cervical muscles. The former is usually, however, preceded by tic of the facial muscles, in particular of the orbicularis palpebrarum. It must be remembered that *facial spasm* may be combined with *spasm of the cervical muscles* without leading to the development of general tic. In such cases a definite diagnosis can only be made after long observation of the condition. As soon as systematic movements or echolalia, etc., appear, the diagnosis of general tic may be given. The two affections are certainly very closely related, and cannot always be sharply differentiated. Myoclonia may involve the muscles of the neck, but is not limited to these, and does not as a rule produce marked movements in them.

The condition is easily distinguished from simple *chorea*, but it may be combined with or followed by this disease, as I have seen. Meige and Feindel have recorded one such case.

Spasm of the muscles of the neck may be a symptom of hysteria, but Kollarits[3] goes much too far in simply classing torticollis as a form of hysteria; we must decidedly differ from this opinion.

Pathology.—So far as we know, there are no changes in the central or peripheral nervous system. The disease is probably due to a *condition of irritation* in the *nerve centres*. I think it probable, as already stated, that the cortex and the zones which include the (kinæsthetic) centres for the cervical muscles are in most cases the starting-point of the disease, and that instability of these centres, mostly inherited or congenital, is the cause of the spasms. Others regard the nuclei of the cervical muscles or even the cerebellum (?) and Deiters' nucleus with its conduction tracts as the site of the affection. Our explanation of the condition, which agrees with that of Brissaud, viz., that it is a psychomotor disease or a movement of expression which has become imperative, applies to the majority of cases.

[1] *Nouv. Icon.*, xviii. [2] *Journ. de méd.*, Bordeaux, 1905. [3] *Z. f. N.*, xxix.

Babinski's view (with which Heldenbergh [1] agrees), that it is due to a condition of irritation in the pyramidal tract, seems to me to have no foundation.

The *prognosis* is generally *grave*. Spontaneous recovery only occurs in mild cases. I have seen the spasms disappear under treatment in a good many cases. Thus a rotatory tic, produced by influenza in a non-hysterical woman, was cured in a few months. The disease usually lasts for years, or even for a whole lifetime; it attains a certain degree of severity and then remains stationary or varies in intensity. It is only in a small number of cases that the condition becomes steadily worse, so that we can use the term spasmus progrediens (Lukàcs [2]). The spasm of the muscles of the neck which develops in early childhood, requires special consideration. Recovery from it is not unusual; indeed, it seems to be the rule in typical cases, and chiefly to follow cutting the teeth (Henoch, Dickson). On the other hand a relationship has been observed in a number of cases between this form of spasm and epilepsy, idiocy, and grave, fatal, brain diseases (meningitis, syphilis ?) (Hochhalt, Böhr, Jacquet). It is open to question, however, whether these were really cases of simple nodding spasm. In those which show hysterical symptoms, complete recovery may take place, and I have also seen this happen after some years in a few severe cases of spasm of the muscles supplied by the spinal accessory, and under such different forms of treatment that I was led to assume the existence of some mental factor. This is in accordance with the views and experience of Brissaud and his pupils, and with my own opinion, already stated, of the nature of this disease.

The condition may, on the other hand, become so distressing as to lead to attempts at *suicide*.

Treatment.—If the cause of the affection can be discovered, treatment must be directed to its removal. Special diet, iron, hydrotherapy, etc., may therefore be prescribed. It is also important to relieve any pressure from the clothing, such as an uncomfortable collar (Marie). The patient should be carefully protected against excitement; he should be exposed as little as possible to the observation of strangers, and should live in a quiet place, either alone or with some trustworthy companion. If alcoholism or any other poisoning be the cause, he should, of course, be removed from its influence. Occupation which does not fatigue him is usually beneficial.

Preparations of bromide as a rule alleviate the spasm, and *morphia* and *opium* are still more efficacious, but these drugs should only be prescribed (and preferably in subcutaneous injections) as a last resort. Curare is said to have been used with success in a few cases. Nerve sedatives may be tried. Tincture of gelsemium, valerianate of zinc, recommended by Erb and Gowers, and extract of conium have been thought to be of service. Romberg advises oxide of zinc for the nodding spasm.

Supporting apparatus may be of use, either in the form of a pasteboard collar round the neck or as a ring round the head, to which is fixed a sort of fork supporting a small pad. The purpose of this pad is to press the head in the direction opposite to that of the spasm. Prolonged pressure upon pressure-points has been used in a similar way. Tight bandages must be absolutely avoided.

In one of our cases warm sea-baths led to complete recovery.

[1] *Belg. méd.*, 1902. See also Beduschi-Bossi, abs. *R. n.*, 1904. [2] *C. f. N.*, 1906.

Electricity has had excellent results in a few cases. If pressure-points can be found—which is not usually the case in this form of spasm—the *anode* should be applied to them. If not, the anode should be placed over the *spinal accessory* nerve, the cathode being placed upon the muscle. *Faradisation* may also cure the spasm. French writers advise its application to the sterno-mastoid of the opposite side, which is usually atrophied. Vigouroux and Charcot have found this successful. We have no definite reports as to the value of *massage*, but Hoffa [1] thinks it should always be used as an accessory method.

Gymnastics, viz., systematic exercises in fixing the head, *e.g.* by the perimeter, as advised by Brissaud,[2] Feindel,[3] Popoff, and others, or according to the "inhibition-therapy" methods which I have recommended,[4] form an important means of treatment. Favourable results have been reported by Brissaud, Meige-Feindel, Fornaca, Cruchet, Pitres,[5] and others. (For an account of these and Pitres' method, see the chapter on general tic.)

As the *mental influence* is an essential factor in most of these methods, one might expect recovery to be also induced by means of pure *psychotherapy*, or hypnotism. I have never found the latter of any use, but others have. Renterghem,[6] for instance, reports having cured a case of rotatory tic by mental and suggestive influence. Isolation may be necessary.

Improvement and even recovery has in a few cases been brought about by the use of *counter-irritants*, the application of a *fly-blister*, a *seton*, and especially by the application of a *button cautery* to the nape of the neck (Busch [7]).

The results of *operative treatment* are doubtful. Section of the tendons of the muscles affected by the spasm has occasionally done good, even in cases other than congenital wry-neck. Since Strohmeyer's first attempt, this method has been very often employed; Kalmus collected ninety-five cases some years ago. Although the operation fell somewhat into discredit on account of some unsuccessful cases, it is again gaining in favour, thanks chiefly to Kocher (see below). *Section*, *stretching*, and even *resection* of the spinal accessory nerve, or neurectomy, very seldom lead to permanent cure. The latter is most likely to be successful. Petit, Babinski,[8] and others have reported cure after these operations, but the time of observation has usually been too short. Francis [9] saw section of the accessory nerve result first in improvement, followed by relapse with mental disturbance, and finally by recovery. In a few cases the superior cervical nerves were sectioned in addition to the accessory, followed apparently by recovery (Smith,[10] Schede,[11] Richardson-Walton, Wölfler [12]) Richardson-Walton recommend resection of the spinal accessory as the first step, the operation being repeated on the superior cervical nerves only if the first operation prove unsuccessful. Risien Russell [13] has studied this question experimentally. I have in several cases seen the spasm,

[1] "Die Orthopädie im Dienste der Nervenheilkunde," Jena, 1900.
[2] "Leçons," etc.; also *R. n.*, 1895 and 1897.
[3] *Nouv. Icon.*, 1897, and Meige-Feindel, "Les Tics," etc.
[4] *Therap. Monatsh.*, 1899, and *Journ. f. P.*, i.
[5] *Journ. de Méd. de Bordeaux*, 1904.
[6] *Psych. en neurol. Bladen*, 1897.
[7] *B. kl. W.*, 1893.
[8] *R. n.*, 1907.
[9] *Lancet*, 1893.
[10] *Lancet*, 1892 and 1893.
[11] *Handbuch von Pentzold-Stintzing*, viii.
[12] *Prag. med. Woch.*, 1900.
[13] *Br.*, 1897.

when its path to the originally affected muscles is blocked, pass over to other muscles. The *central irritation* therefore remains and finds its discharge by other channels. In one of my cases section of the tendons was followed by stretching, then by section and resection of the accessory nerves. The result was that, notwithstanding complete atrophy of the sternomastoids and incomplete atrophy of the trapezius, the spasm became even more intense, now involving the splenius, the omohyoid, and the remainder of the trapezius. I then applied a seton, but without avail. This was followed by cold-water treatment, which was most successful. F. Krause has removed the spinal accessory in several of my cases. The direct result was very slight, but subsequently, at least in one case in which the tendons had also been sectioned, considerable improvement took place. In these cases the trouble was particularly severe and extensive, and it need not be said that all the milder methods had previously been tried.

These operations probably owe their effect for the most part to their action as *counter-irritants*, and their influence upon the mind. Several cases have, however, come to my knowledge in which, after counter-irritants of every kind had been used in vain, recovery was induced by an operation performed by Kocher of Bern, which, according to the description of the patients, consisted of *section of the tendons of almost all the muscles of the neck*. The operation has since been described by Kocher [1] and Quervain. The sternomastoid, trapezius, splenius, complexus, and inferior oblique were all divided. The operation was not performed at one sitting. The after-treatment consisted of gymnastics. Seven out of twelve cases were cured, but recovery does not always seem to have been complete. Other surgeons, *e.g.* Nové-Sosserand,[2] Ebers,[3] and Manasse, have been successful with this operation.

I have recently met a few cases in which Kocher's operation was unsuccessful. Section of the second cervical branch combined with division of the contracted cervical muscle is recommended in torticollis by J. Berg (*Nord. med. Ark.*, 1905), but he would only resect the spinal accessory nerve if the sternomastoid is severely involved.

Brissaud is opposed to surgical treatment, and especially to Kocher's operation, on account of the psychogenic nature of the disease. Sicard-Descomps [4] also report failure of operation. In any case surgical treatment should only be resolved upon if the other methods have proved unsuccessful.

Coudray also prefers slighter operations, such as resection of the external branch of the spinal accessory, to Kocher's method, as he regards the mental influence as the essential factor. Walton (*Journ. Nerv. and Ment. Dis.*, 1897) strongly approves of surgical treatment. Collier (*Lancet*, 1900) compresses the exposed accessory nerve with a silver-wire ligature. Corning (*N. Y. Med. Journ.*, 1894) recommends a peculiar operation, which he calls *elaeomyenschisis*: he injects into the affected muscle an oil which becomes solidified when cooled (mixture of ol. theobromini and paraffin), and solidifies the mixture with an ether-spray (!).

Injections of atrophin into the muscle, continued for a considerable time, are said to have been of service in a few cases (Potts, Lesczynsky).

When all is said, however, treatment of this condition rests upon very unsatisfactory grounds.

[1] See the description of the method in Kocher's "Chirurg. Operationslehre," fifth edition, 1907.
[2] *Lyon méd.*, 1898. [3] *A. f. P.*, xxxvi. [4] *Nouv. Icon.*, 1907.

Spasms of the Muscles of the Trunk and the Extremities

If we exclude the various organic diseases of the brain and spinal cord which give rise to spasm of the muscles of the trunk and extremities, and the motor neuroses in which there is spasm of all the muscles of the body or of those employed in certain occupations, we may consider *idiopathic* and *localised* spasm in the muscles of the trunk and extremities as an uncommon symptom. It has, nevertheless, been observed, though only in rare cases, in every muscle of the body.

It may affect a single muscle, such as the *rhomboid*, the *levator anguli scapulæ*, the *latissimus dorsi*, the *deltoid*, or the same muscles on both sides of the body, *e.g.* the *pectorales*, etc., or a number of muscles in various combinations, *e.g.* the levator anguli scapulæ along with the rhomboid, the triceps with the pectoralis major, etc. The spasm may sometimes involve a *group of muscles* innervated by the *same nerve* or by one pair of roots. Thus I have seen clonic twitching in the *deltoid, biceps, brachialis internus,* and *supinator longus*, which followed in the same order as that produced by electrical stimulation from Erb's point. In a case treated by Laquer (*A. f. P.*, xxi.) the clonic twitching was limited to the muscles innervated by the musculo-spiral nerve; in another (Hochhaus, *D. m. W.*, 1898) the triceps and supinator longus were alone affected. Schultze (*A. f. P.*, xxi.) describes a triceps spasm, and Erben a triceps clonus which appeared when the forearm was flexed. Clonic spasm in the muscles of the forearm, especially in the pronator teres, was observed by Bernhardt (*A. f. P.*, xix.). Féré (*Rev. de Méd.*, 1894) describes spasm limited to the muscles of the little finger, and Schultze (*Z. f. N.*, iii.) tonic spasm in the muscles supplied by the ulnar nerve.

Tonic spasm has been frequently observed in single muscles of the shoulder (rhomboid, levator anguli scapulæ), in the flexors of the fore-arm, the flexors of the hand and fingers, and the interossei. It often develops into contracture. Weir Mitchell mentions a case in which spasm of the flexors of the fingers was so intense that the nails pierced the palm of the hand.

Of the muscles of the lower extremities, the calf-muscles most often show painful tonic contraction of short duration (cramp). Such a spasm also occurs in the *extensors of the foot and toes*, the *plantar muscles*, the tensor fasciæ latæ, quadriceps, ileopsoas, etc. In a congenitally nervous patient of mine, for instance, tonic spasm developed in the *right psoas* and *sartorius*, which lasted for half an hour and occasionally extended to the *rectus femoris* and *gracilis*. A physician who suffered from this condition came to consult me, fearing that he had Thomsen's disease.

Such muscular spasms are often due to poisoning and errors of metabolism; they often occur, *e.g.* in alcoholism, diabetes, lead-poisoning, cholera, typhoid, diarrhœa, after long marches, etc.

In one case I could only attribute them to improper use of a hair-dye. They may also be due to the neuropathic diathesis (occurring in the course of hysteria, etc.).

Of the later papers on this subject see those of Féré, "Les Crampes et les Paralysies nocturn.," *Méd. mod.*, 1900, and Naecke (*N. C.*, 1901, and *M. f. P.*, xx.).

Muscular spasm may in rare cases involve more or less every muscle of the body. Wernicke (*B. k. W.*, 1904) gives to this form the name of "*cramp-neurosis.*" See also Bechterew (*M. f. P.*, xvii.).

In one case in which the tensores fasciæ latæ, the extensors of the thigh and the recti abdominis had been affected for six years, the muscles became markedly *hypertrophied* (Schultze). Berger (*N. C.*, 1882) has described tonic spasm of the *cremasters*.

Tonic and clonic spasms have several times been observed in the quadriceps; in one case they only appeared in walking. The adductores femoris and the rotators seem to be much less often affected, but Bernhardt has described clonic spasm of the muscles of the thigh which involved the adductors. We know little for certain as to spasm of the flexors of the leg. Spasm of the calf-muscles has been found as an occupation neurosis in a harpist by Bonnus (*R. n.*, 1904).

Rhythmical *clonic twitching* was in one case limited to the *psoas-iliacus*, extending later to the opposite side. In another case (Jobert) the *peroneus brevis*, and in a third (Bernhardt, *B. k. W.*, 1893) the *peroneus longus and brevis* were involved. In the latter the spasm persisted during sleep. Movement of the tendons in the groove behind the external malleolus produced a crepitating sound. Manipulations which for a time inhibited the spasm (compression of the tendon, stretching the muscle, etc.) caused it to pass over to other muscles. Clonic spasm occasionally also occurs in the tensor fasciæ latæ. This group possibly includes the muscle waves (*myokymia*) described by Kny (*A. f. P.*, xix.) and Schultze (*Z. f. N.*, vi.), which consist of prolonged and marked fibrillary tremor and undulation of the muscles, especially in the legs. These may be painful, and may be accompanied by hyperidrosis. Increase of the electrical excitability has

also been noted. Chill, trauma, lead-poisoning, and exhaustion were the causes. Cases of this kind have also been published by Hoffmann (*N. C.*, 1895), Meyer, Karcher, Ballet, Newman, Grawitz-Meinertz (*N. C.*, 1904). I have treated a patient with neurasthenic melancholia in whom myokymia of the calf-muscles had been present from birth (stigma ?), and was accompanied by weakness or absence of the Achilles-jerk.

A very peculiar form of spasm, limited to the *tunica dartos*, resembling in its nature the so-called scrotal reflex, occurred in one of my neuropathic patients.

The majority of those who suffer from such spasms are exceedingly *nervous*. Signs of hysteria are present in many cases, or conditions or attacks of a hysterical character have previously been present. In others there is *neurasthenia* or *psychasthenia*, or a marked neuropathic predisposition. In one case there had been *epilepsy*, in another *writers' cramp*, in a third *chorea*, and so on. This predisposition is, however, often absent in cases of tonic spasm, which corresponds to cramp, and is more often due to *over-exertion*, *local* irritation (varicose veins), or *poisoning* (alcoholism, diabetes, cholera, etc.).

Trauma is also an important factor in the etiology. The spasms are either the direct result of injury or are caused by sensory irritation produced by the wound or cicatrix. The trauma has this effect in congenitally nervous individuals, or it may have a simultaneous action upon the brain, producing in it the abnormal excitability which responds to stimulation from the periphery accompanied by spasm. In the case published by Laquer, energetic massage used for writers' cramp had the effect of a trauma. Local muscular spasms have frequently followed *inflammation of a joint*, and there can be no doubt as to their *reflex* origin. This view is in accordance with the fact that neuralgia and pain arising from the *stump of an amputation* may give rise to local spasms. Although the nervous system when quite healthy does not react in this way to pain, prolonged continuance of the pain may produce the central diathesis.

The *prognosis* as to recovery is doubtful. Ordinary cramp in the calf-muscles is of course quite a simple condition which as a rule disappears spontaneously, but it may be very persistent. All these spasmodic conditions may be exceedingly obstinate, lasting for months or even for ten years, but as a rule they sooner or later suddenly disappear.

Treatment.—The remedies which have proved efficacious are of many kinds. Some cases yield to electricity, a single application of the anode, or longer use of the constant or the galvanic current—others to gymnastics, especially inhibition-exercises, cold douches, tenotomy of the muscle, or to the mere threat of operation—in short to the most *heterogeneous methods*. This fact is an indication of the *psychogenic* character of the spasms in many, possibly in most cases, and of the power of suggestion. It is not always successful, however, nor is it certain that all the remedies act in this way. *Local massage*, *wet packs*, *warm baths*, alcoholic rubbing, *irritant ointments*, and *sinapisms* may all be used. Schleich's injections may have a good effect. It is important for the patient to avoid all over-strain. A spasm in the muscles supplied by any nerve may sometimes be alleviated by galvanism of that nerve.

It is obvious that the general nervousness which is usually present calls for appropriate general treatment.

Respiratory Spasm

Tonic spasm of the diaphragm is very uncommon. In it the epigastrium is protruded. The clear resonant note on percussion over the lungs extends low down, abdominal respiration is absent, and the upper parts of the thorax are set into motion by the rapid, dyspnœic breathing. The patient has a feeling of suffocation, and usually pain at the insertions of the diaphragm. The diaphragm does not move during respiration, and so-called acute emphysema of the lungs may result from this spasm.

The condition may be due to *hysteria*, and may disappear quickly and spontaneously or persist for a long time in a milder form. In other cases it becomes a grave disease, giving rise to great anxiety and leading to marked asphyxia. Such cases should be treated by *cold douches*, the patient being in a *warm bath*, *hot fomentations to the epigastrium*, *faradisation*, *treatment of the phrenic by the stabile anode*, and possibly *morphia* or even *inhalations of chloroform*. The spasms of tetanus and tetany may also affect the diaphragm.

Clonic spasm of the diaphragm (*singultus*, *hiccough*, etc.) is much more common, as everyone knows from personal experience. Sudden contraction of the diaphragm gives rise to an *inspiratory sound*, as the glottis is not simultaneously dilated. As a rule the muscles of deglutition

apparently also become contracted. In mild cases, in which the spasmodic movements are not severe or frequent, the person (we can hardly say patient) is not really greatly inconvenienced. In severe cases with very forcible inspiration, the other respiratory muscles are usually involved; a hundred or more spasmodic movements may occur per minute, and the condition is very distressing, rendering speaking, eating, and breathing very difficult. The chief danger is, however, that it may become established and persist for months or even longer.

The *singultus* of *hysteria* may become equally intractable. It is in rare cases an ominous symptom of *organic brain disease* (apoplexy, tubercular meningitis), appearing just before death. It may possibly be caused by *direct irritation of the phrenic nerve*. Thus Strümpell mentions a case of mediastinal pericarditis characterised by this symptom. The spasm is undoubtedly sometimes produced by irritation from the *genital organs* or the *gastro-intestinal tract*, but it may be caused by emotion, and mental infection is an important factor (Bernhardt).

Other forms, such as *spasmodic yawning* (oscedo, chasma), *spasmodic sneezing* (ptarmus, sternutatio convulsiva) are hardly in themselves diseases; they occur from time to time in hysteria and in nervous persons, but are practically never of distressing severity. Spasmodic yawning may form the aura of an epileptic attack, and it is sometimes observed in organic brain diseases, especially in tumours and abscesses of the cerebellum.

Some time ago a woman suffering from general neurofibromatosis, consulted me on account of spasmodic *snoring* (ronchospasm); she was constantly forced to produce the well-known sounds emitted by many people when fast asleep. The psychogenic origin was shown in this case by the fact that the snoring stopped when her attention was distracted. Treatment was of some use, but I have lost sight of the patient.

Singultus may sometimes be arrested by holding in the breath, by straining, distracting the attention, fright, etc. If it is very persistent, *counter-irritants* (cantharides, mustard plaster, or faradisation applied to the pit of the stomach, etc.), or sedative drugs (bromide of potassium, opium, chloroform, etc.) may be tried. I have occasionally found bismuth. subnitr. of service. Laborde and Noir (*Prog. méd.*, 1900) recommend rhythmical traction on the tongue, which I have sometimes found useful. Faradic and galvanic treatment of the phrenic nerves, passing a sound down the œsophagus, compression of the lower parts of the thorax, and many other methods have been recommended. Psychotherapy is evidently the chief factor in the cure.

The spasms which affect some or all of the muscles of respiration and produce rapid, forced, respiratory movements, are almost always of a *hysterical* nature (*q.v.*). A very peculiar case which I had an opportunity of seeing has been described by Edel (*B. k. W.*, 1895): A man suffered for many years from respiratory spasm, which was brought on by any touch, noise, light, etc., and was accompanied by severe dyspnœa. No other symptom could be discovered except an increase of reflex excitability. Nervous tachypnœa also occurs in neurasthenia and exophthalmic goitre. Unilateral clonic spasm of the respiratory muscles is described by Schapiro (*Z. f. k. M.*, viii.) A pure unilateral or bilateral expiratory spasm caused by spasmodic action of the abdominal muscles may also occur.

Spasmodic attacks of coughing (except those of laryngeal crises) are usually hysterical. They may have a reflex origin from the abdomen, the external ear, the nose, and possibly even from the liver and spleen.

Saltatory Reflex Spasm [1] (Static Reflex Spasm)

Saltatory reflex spasm is the name given to a peculiar form of spasm of the lower extremities first described by Bamberger. As soon as the patient puts his feet to the ground, he begins to make springing, hopping, dancing movements, which are caused by clonic contractions in the muscles of the lower extremities, especially those of the calves. In milder cases the patient may stand on tip-toe, whilst his heels move spasmodically up and down. The spasm disappears when he assumes a recumbent position, but in many cases it may be brought on by touching the soles of the feet.

There are either no other symptoms, or merely those of general nervousness and hysteria. The cutaneous and tendon reflexes are usually increased, and it has been thought that the latter symptom is of special importance (Erlenmeyer, Kast).

[1] For the literature, see Erb, "Krankheiten des Rück.," etc., 1868, Abt. ii.; and Bernhardt, *Nothnagels Handbuch*, xi., second edition, 1904. Later contributions to the subject have been published by Decroly (*Journ. de Neurol.*, 1904), but his views are open to objection.

The affection occurs in both sexes and at any age, and it may have a spontaneous onset or follow an *infective disease*. It is probably not an independent form of spasm, but merely a symptom or a rare form of *hysteria*, psychasthenia, or tic. It may occur as an occupation neurosis in ballet-dancers.

The *prognosis* is favourable. After lasting for some weeks or months, the spasm usually disappears. In one case it is said to have persisted for years until the patient died. Sedatives, electricity, wet packs, anæsthetising of the soles of the feet, etc., may be employed, and above all psychotherapy. Gowers recommends diaphoretic treatment.

Localised and General Tic (Tic Général, Maladie des Tics)

ERINNERUNGSKRÄMPFE (FRIEDREICH). MALADIE DES TICS IMPULSIFS (MARINA-JOLLY). MYOSPASIA IMPULSIVA

The term *tic* is adopted from the French. The originally vague conception of this condition has lately become more and more sharply defined. The condition has, however, been known for a very long time. It was certainly recognised by Friedreich, and especially by Trousseau. Charcot has discussed it several times, and to his pupils, Gilles de la Tourette (*A. de Neurol.*, 1885) and Guinon (*Rev. de Méd.*, 1886) in particular, we owe a definition and excellent descriptions of the disease in its fully developed form. My own studies and contributions to this subject extend back almost to the time when these writers published their fundamental work. I would refer to the dissertation of Färber, written under my direction (Berlin, 1885), and of O. Müller (Berlin, 1889), and to my papers in the *B. k. W.*, 1887 and 1889. Further advance was made by the work of Brissaud, and especially of his pupils Meige and Feindel. Their work, "Les Tics et leur Traitement" (Paris, 1902) (English translation by S. A. K. Wilson, London, 1907; German by O. Giese, Leipzig, 1903), is an excellent monograph upon this affection. I have already indicated in the second edition of this text-book that analogous or allied conditions may occur in animals, particularly in horses. The affection has long been known to veterinary surgeons. French neurologists (Meige and Feindel, Rudler-Chomel, *Nouv. Icon.*, xvi.) have only recently devoted careful study to these animal tics (tic de l'ours, tic de léchage, etc.).

Tic is a reflex, defensive, or voluntary movement, which has assumed an imperative character. Although the motor action may in itself resemble one of these movements, *e.g.* reflex closing of the eyes, grimacing, angry clenching of the fist, the gesture of amazement, etc., its pathological nature is shown by the fact that it is not called forth by any external excitation, and therefore by an adequate mental process, but is due to some incontrollable motor impulse. The memory image of the motor process is excessively vivid, and the patient is irresistibly compelled to carry it into actual realisation. He can oppose the imperative impulse to a certain extent, but his power to inhibit it is limited, and the effort to control it gives rise to a feeling of distress which seeks its discharge in the motor act. Tic is further distinguished from simple reflex movements or movements of expression by the short, abrupt, and forcible nature of the movements, and by their constant or frequent repetition.

The trouble may consist in a single movement,—an *isolated tic*,—in a number of different movements which take place at the same time or in rapid succession, or in a *general* tic. It is impossible to differentiate sharply between local and general tic, as they may blend into each other.

Some of the localised tics, *e.g.* convulsive tic of the facial and cervical muscles, have been described in the foregoing chapter, but tic may be confined to any other individual group of muscles. As the definition implies, there is usually a combination of various movements involving muscles innervated by different nerves. Thus, we have a "sucking-tic, a snuffing-tic, a licking-tic, a biting-tic, a grinning-tic, a scratching-tic, a twitching-tic, a nodding-tic, a gulping-tic," etc. It has to be borne in

mind, however, that the purposeful and intentional character of the movement is not always clearly evident, as in addition there may be simple twitchings which cannot be regarded as pathological gestures.

The "*maladie des tics convulsifs*" of Guinon and Gilles de la Tourette is characterised, according to these authors and to our own observations, by the following symptoms: 1. twitching of the facial muscles; 2. systematic movements, constantly repeated in the same way; 3. echolalia and copralia, and perhaps echokinesis, and sometimes by 4. fixed ideas and imperative actions.

The disease usually develops in *children* of the age of seven to fifteen, who have a *hereditary* predisposition. As a rule the neuropathic tendency is a general one (hérédité polymorphe). I have only been able to discover direct heredity in a few cases. In one, the grandmother of the patient had had a tic which she had transmitted to four of her daughters and three of her grandchildren. One of the patient's brothers was epileptic. Direct heredity is reported in a few cases by French authors (Gintrac, Letulle, Charcot).

The first symptoms usually follow some mental excitement, or they may be produced by trauma, over-strain, or an infective illness. Meige and Feindel think the disease is caused or brought on by improper upbringing and by mental infection (imitation). The first sign which strikes the parents is usually *twitching of the facial muscles*, in particular blinking of the eyes, contortion or rapid opening and shutting of the mouth, etc. These are followed by twitching of the *neck muscles* (sterno-mastoid, trapezius, etc.). Later, perhaps only after some years, movements appear which give an impression that the patient is carrying them out *for a certain purpose*; to express an *emotion*, or as the result of a *habit*, and that the movement is therefore an intentional or reflex one. The patient may touch his nose, stroke his chin or his beard, lift up his collar, throw his head from side to side, catch imaginary insects, expectorate, clap his hands, stamp his feet, open his mouth wide, show his teeth, or imitate dancing, springing, hopping, etc. These movements are repeated in a stereotyped manner. The patient is constantly compelled to begin the same muscular activity over again, and the movement, originally perhaps purposeful and physiological, now becomes pathological. The muscles of the face, neck (especially the sternomastoid), and upper extremities are chiefly affected, but any muscle may be involved. The tic of the eye muscles, described by Lerch, Meige, and Feindel,[1] is very rare. The muscles on the two sides do not as a rule act symmetrically.

The muscles of articulation, phonation, and respiration are mostly involved. The patient feels compelled to utter inarticulate sounds or words. There is often simple *sniffing*, *smacking* of the lips or hissing, still more often *imitation of the calls of animals*. Some patients utter senseless words (one of my patients constantly repeated: Kritsch, kritschkratsch, quatsch, krum dum krikideidei), or words with some horrible, obscene meaning (*coprolalia*), such as "ass, swine, hold your tongue," or terms of a sexual nature. In rare cases there is an impulse to repeat words or sounds (*echolalia*), or to imitate movements (*echokinesis*).

Meige and Feindel have rightly pointed out that tic may take the form

[1] *Loc. cit.* and *Ann. d'oculist*, 1903.

of tonic muscular spasm, of an imperative attitude (of the head or arm). Rudler [1] also reports a case which he explains in this way.

The patient, whose intelligence is usually unimpaired, suffers greatly from these imperative movements, especially if he is advanced in years. Many succeed by a strong effort of will in suppressing the movements for a time, but few can control them for very long. A ballet-dancer, for instance, suffering from tic, was able to appear in public every evening; a post-office official under my care could perform his duties without attracting the attention of those around him, but this repression of the impulse causes an internal feeling of discomfort and anxiety, and the tic subsequently becomes all the more imperative. This repression of the movement during systematic occupation is produced not merely by self-control, but by the *distraction* or *concentration of the attention*. If the patient is alone and free from observation, he is often able to work without being disturbed by the muscular twitching. But when he resumes his contact with the outer world, and notably when he becomes excited, the muscles at once become active, the twitching corresponding in intensity to the measure of his excitement. One of my patients said he felt compelled to speak a word as soon as it entered his mind, and the harder he tried to repress it, the more distressingly urgent the impulse became.

Voluntary movements as a rule have a *soothing* effect upon the tic, but if the patient is very much excited, or if the imperative actions have reached a certain stage of intensity, these may for a time become themselves weakened.

The intensity of the spasmodic movements varies greatly; complete *remissions* and paroxysmal exacerbations may occur.

In some cases the patient is dominated by *morbid ideas*—not hallucinations, but *imperative ideas* with corresponding imperative actions, *e.g.* the impulse to count (the windows of a house, the number of steps, etc.), to continue touching certain objects, or, after having taken a certain number of steps forwards, to take so many backwards.

One of my patients felt compelled to draw out the concluding *r* of a word; he said father-r-r-r; another could not resist the mania for collecting bits of paper, food, etc., which he carefully gathered into little bags: when one was full, he procured another. The same patient stood for hours before a glass, combing his hair without ceasing.

The person with a tic is certainly a neuropath or a psychopath, but he is not suffering from a mental disease. He has not even any tendency to insanity, although a psychosis may develop from the same foundation. The memory, also, frequently becomes impaired as the result of the persistent demands made upon the attention. Absent-mindedness and inability to concentrate the attention are almost constant symptoms of this disease, a factor which makes the school-training of a child with tic a peculiarly difficult task. In one of my patients this inattention was so marked that for a time he gave those round him the impression of being deaf. The close connection between tic and imperative ideas is shown by the fact that one member of a family may suffer from the former, another from the latter (example: a father has *folie du doute*, a daughter general tic, etc.).

Meige and Feindel lay stress upon the weakness and inconstancy of the will-power, which gives an infantile stamp even to the character of adults

[1] *Nouv. Icon.*, 1903.

suffering from tic. From the nature of the disease, it is to be expected that the patient will often isolate himself from society and thus accentuate his peculiarities.

Bresler (*N. C.*, 1896) regards tic as a motor reaction to an originally mental shock, therefore as a kind of psychical defensive movement or spasm of expression. G. Köster (*Z. f. N.*, xv.) thinks it is due to absence of inhibition caused by exhaustion of the co-ordinating centres which regulate the functions of the lower neurones (compare my remarks on p. 1249).

Course and Prognosis.—The condition is a *chronic* one, which lasts for years or for a whole lifetime. Many authors regard it as incurable, but quite erroneously. I have seen typical cases completely *recover*. In one girl, who had had a general tic from the age of four or five, complete recovery coincided with the onset of her periods, and long intermissions had previously been brought about by treatment. One man who brought his son suffering from tic to consult me, had himself been under my care for the same condition thirteen years previously, and had been absolutely cured (see my paper on the prognosis in the *Journ. f. Psych.*, i.). I have treated two brothers for a very severe form of tic dating from childhood. One was practically cured, and the other hardly improved at all. In some cases the condition is chronic or progressive. It is naturally very intractable in imbeciles and idiots (Meige). Self-inflicted injuries, *e.g.* biting the tongue, plucking out the hair, etc., are rare incidents in the disease.

The *diagnosis* of the tics is often a difficult matter. Fully developed cases can hardly be mistaken. If the twitching is limited to the facial muscles, it may be mistaken for facial spasm ; see, however, p. 1239. If the spasm extends to the muscles of the neck, the diagnosis is made probable, but is only confirmed by the appearance of systematised tics. (There are also combined spasms of the muscles of the face and neck which do not strictly belong to this class, although they are closely related to it.) Some patients exhibit a certain tic, such as clearing the throat, shrugging the shoulders, sniffling, snapping the fingers, introducing some meaningless word into his speech, etc., unaccompanied by any other symptom. This may be a case of *habit* or of localised tic. If it is the former, the spasmodic, forcible, irresistible character of the movement and its tendency to become general are absent, as Séglas, Letulle, Meige-Feindel, and Dromard [1] have rightly pointed out. There is no justification for speaking of such cases as an abortive form of the *maladie des tics*, the less so as this tendency to the habitual carrying out of some movement exists in many children, and can often be overcome at a later period by voluntary effort.

This habit-spasm is specially discussed by English writers, *e.g.* B. Still, *Lancet*, 1905.

There are of course other cases in which the condition is so slight and runs so protracted a course that a " localised tic " appears only from time to time, and the symptoms which *co-exist* in severe cases are to a certain extent dissociated and only come to full development after many years. Thus one patient consulted me for eructations, which hardly ever occurred except at night. I happened once to be a witness of this symptom, which he described as an " eructation," and I saw that it was not a true eructation, but a spasm of the muscles of articulation and voice production, undoubtedly of a psychogenic nature. Further investigation

[1] *Journ. de Psych.*, 1905.

showed that he had from childhood contracted the habit of crossing his legs from time to time in walking, or of rubbing one leg against the other, and that at other times there was twitching of the muscles of the neck. He also occasionally suffered from fixed ideas. Heredity was at first denied, but shortly afterwards I had an opportunity of treating the father of the patient for melancholia, and of learning that he had previously suffered from a mental disorder of the same kind.

There is a *mild, slight, comparatively benign*, and also a *severe* form of general tic. The former is a very common affection.

I have met with a few cases which were difficult to classify. Thus, a boy was sent to me who suffered from nocturnal and diurnal enuresis. His parents stated that at night during his sleep he made rhythmical movements of the head and trunk, singing a melody as he did so. Whilst he was awake during the day, he showed no abnormality except a slight blepharoclonus, and I was satisfied that the case was an atypical one of general tic. I might mention (1) that I have often, in contrast to Meige, observed the symptom of a *nocturnal* or somnolent occurrence of these motor neuroses (compare p. 1250 and the chapter on chorea); and (2) that I have repeatedly found the symptom of *involuntary micturition* in patients with general tic, and have ascertained it to be merely a manifestation of the primary affection. The idea of micturition immediately provoked the motor act, or the corresponding sensation was at once translated into action because the power of inhibiting it was impaired, or, finally, the micturition might be due to absent-mindedness, already mentioned as a characteristic of these patients.

This symptom has recently been studied by Meige (*R. n.*, 1904; *Journ. f. P.*, ii.). His reference to an earlier communication by Brissaud is not, however, correct, as in it the incontinence which occurs in tic has not received any consideration.

The condition differs from *chorea minor*, for which formerly it was often mistaken, in the *systematic* character of the movements, the comparatively lengthy *pauses*, the soothing influence of work, and the echolalia and coprolalia. The *maladie des tics* may apparently be combined with chorea (Oppenheim, Raymond), as there is a marked tendency of the various forms of spasm to be associated with each other. *Hysteria* may give rise to spasmodic symptoms very like those of general tic. These come on suddenly, however, generally after some excitement or a convulsive attack, and are associated with the stigmata of hysteria. The echolalia and copralia are also generally absent, and the affection may be influenced by suggestion, but the diagnosis is not always certain. In fully developed cases, tic can be distinguished at the first glance from *paramyoclonus* (see following chapter), but some writers are inclined to regard these two conditions as identical. It is probably identical with, or closely related to the conditions of jumping in America, of latah (in Malay), of meriatschenje (in Siberia), and of imubacco (Sakaki [1]).

It should be remembered that there is very often at the onset and in the course of dementia præcox a tendency to grimacing, and that imperative movements and actions have also been observed in the course of *paranoia*; these, however, are caused by hallucinatory ideas.

Treatment.—Sedatives and nerve tonics have usually no effect, but

[1] *Neurologia*, i.; *N. C.*, 1903.

bromides may alleviate the tic. At the height of the paroxysm it may be necessary to use chloral hydrate and chloroform inhalations, in order to procure a few hours' rest for the patient. Arsenic is of no use. I have occasionally prescribed hyoscin and eserin, but without any result, whilst Rosenfeld found the former of service in one case. The symptoms only disappeared for a few days in one of our cases, in which eserin-intoxication developed. Wagner reports having found thyroidin successful in a few cases.

Mild *hydrotherapy* (especially wet packs), and, above all, *isolation* of the patient may have a soothing effect. I have occasionally seen marked improvement after the application of a seton. In one case tic of the facial muscles disappeared after the removal of enlarged tonsils.

Hypnotism is, so far as our experience goes, practically useless. Most of the patients cannot be hypnotised. A few cases of benefit have been reported by Raymond-Janet, Wetterstrand,[1] and others.

On the other hand, I have found *gymnastic exercises* of great service. I make the patient go through a number of gymnastic movements under trained supervision. These are followed by exercises which consist in keeping the body and each of its parts quite still, in suppressing reflex and emotional movements, etc. These are at first difficult, and should be very short to begin with, and only gradually lengthened. I have found it advisable to follow up this enforced stillness immediately by other active gymnastics (free exercise, etc.).

I have developed these therapeutic measures (*Therap. Monatshefte*, 1899) independently of Brissaud, whose first publications no doubt preceded mine, and of Meige and Feindel. Our experience and advice are identical in many points, but mine include an important factor which is not mentioned by these authors, viz., practice of repression of the *reflex* and *defensive movements, and of the expressions of emotion*. For instance, I make a dash with a sharp instrument at the patient's eye and tell him to repress the lid-reflex; in the same way I make him practise control of the nasal reflex, the defensive movements to painful stimuli, the tickling-reflex, etc., and in this way strengthen his power of inhibition.

Meige and Feindel make their patients drill before a mirror. They speak of *psycho-motor discipline*, I of *inhibition-gymnastics*.

Pitres (*Journ. de méd. de Bordeaux*, 1901) and Cruchet (*Thèse de Bordeaux*, 1902, and Traité des Torticol. spasmod.," Paris, 1907) recommend respiratory exercises, which they have found useful in a few obstinate cases. See also Tamburini (*Riv. sper. di Fren.*, 1903; Porot (*Lyon Méd.*, 1905), and Bruel (" Traitement des Chorées et des Tics," Paris, 1906).

Paramyoclonus Multiplex (Friedreich)

Myoclonia. Polyclonia

Literature: Friedreich, *V. A.*, Bd. lxxxvi.; Schultze, *N. C.*, 1886 and 1897, and *A. f. P.*, xxix.; *Z. f. N.*, xiii.; Marie, *Progrès méd.*, 1886; Remak, *A. f. P.*, xv.; Unverricht, " Die Myoklonie," Leipzig und Wien, 1891; also *Z. f. N.*, vii.; Ziehen, *A. f. P.*, xix.; Erb, *M. m. W.*, 1894; Marina, *A. f. P.*, xix.; Raymond, *Progrès méd.*, 1895, and " Clinique des malad. du système nerveux," 1896; Böttiger, *B. k. W.*, 1896; Lundborg, " Die progr. Myoklonus-Epilepsie," Upsala, 1903; Hunt, *Journ. Nerv. and Ment. Dis.*, 1903; Clark, *Arch. of Neurol.*, 1899, and *R. of N.*, 1907; Dana, *Journ. Nerv. and Ment. Dis.*, 1903; Wollenberg, Nothnagel's " Handbuch," xii.; Mott, *Arch. of Neurol.*, 1907.

This disease was first described by Friedreich in the year 1881. The observations since reported by other writers correspond only in part to the description given by Friedreich.

[1] *L'hypnotisme*, etc., Paris, 1899.

The affection is characterised by *clonic contractions*, affecting mainly the *muscles of the extremities and trunk*, and rarely, if ever, those of the face. The contractions are *short* and lightning-like ; they involve a small number of muscles which *do not have a synergic action*. The effect of the contractions in moving the limb is therefore slight or entirely absent. The contractions involve the muscles of the two sides of the body almost equally ; they may be symmetrical, but not *synchronous* nor rhythmical. The various twitchings, of which there may be sixty to one hundred and more in a minute, are separated by intervals of varying duration. They affect a *single muscle*, which cannot be voluntarily contracted by itself, *e.g.* the *supinator longus*. They may even be limited to parts of a muscle. The clonic spasm may be associated with a fibrillary tremor. Although all the muscles are liable to be attacked, some are involved in almost every case, viz. the *supinator longus*, the *biceps*, the *trapezius*, the *quadriceps femoris*, the *semitendinosus*, etc.

Active movements, which as a rule are unaffected, have a tranquillising, *soothing* effect upon the spasm. Emotion has the opposite effect. The twitching diminishes when the attention is distracted, and disappears entirely during sleep. The tendon reflexes are usually greatly increased. A tap upon the patellar tendon, and even a touch on the skin exaggerates the convulsions.

The phenomena of motor excitement are usually the only symptoms. All the other functions, including the mechanical and electrical excitability, are normal.

I have occasionally observed a combination of paramyoclonus and urticaria. In one case I saw a variety of this disease develop in a woman suffering from progressive muscular atrophy, and also from fear of a railway accident, etc.

In several cases the affection followed fright, in others an infective disease, and in a few it was due to trauma.

Leubuscher (*M. f. P.*, xix.) diagnosed myoclonia in a patient who had to handle lead in his work and who showed symptoms of lead-poisoning.

Unverricht has described a special form of myoclonia which is characterised by its *familial* nature and its association with *epilepsy*. In some of these cases the contractions involved the glosso-pharyngeal muscles and those of the diaphragm. Cases of this kind have since been described by Weiss, Krewer, Sepilli, Lundborg, Clark and Prout,[1] Faber,[2] and Reynolds.[3] Lundborg has devoted very careful study to the condition, which he has found to be very common in the Swedish race.

From the cases that have been published it appears that the epilepsy occurs at first at rare intervals, then more frequently and especially at night, and diminishes in advanced life, when myoclonia, on the contrary, becomes more and more marked and persistent. It affects the voluntary movements, which become gradually more restricted. According to Lundborg's experience the patients have good and bad days, especially during the first stages of the disease. The myoclonic symptoms are increased by the influence of emotion (the psycho-clonic reaction of Lundborg ; he speaks also of a psycho-tonic contraction, *i.e.* tonic contraction of certain muscles during embarrassment and consequent inhibition of movement). The myoclonic symptoms are specially apt to be brought on on the bad days by sensory stimuli (senso-clonic reaction). In Unverricht's type the mechanical excitability of the nerves and muscles is said to be increased.

[1] *Amer. Journ. Insanity*, vol. lix. (abs. in *C. f. N.*, 1903).
[2] *Hospitalstid.*, 1901.
[3] *R. of N.*, 1906.

Lundborg lays special stress upon the development of this condition into *dementia*; indeed, he speaks of *dementia myoclonica*, which he regards as parallel to dementia præcox. I have seen a case of this kind in which the dementia myoclonica was associated with acromegalic symptoms (it has been more precisely described by Graves [1]), and another in which the disease, apparently caused by an injury, was combined with idiocy and atrophy of the optic nerves. I do not feel justified in extending the conception of myoclonia to include this case, and would by recording it merely point to the symptomatic value of the symptom.

Males are chiefly affected.

The difficulty of selecting from the "chaos of the motor neuroses" certain forms, and differentiating them sharply from others, becomes very obvious when we attempt to make good the theory that paramyoclonus is a nosological entity. As soon as we depart from the picture drawn by Friedreich, we run the risk of encroaching upon the region of some other neurosis. Nevertheless it has been doubted whether we are right in regarding Friedreich's disease as an independent one (Möbius,[2] Strümpell, Schupfer,[3] Hartenberg), and many authors are inclined simply to classify myoclonia with hysteria. I regard this attempt as unsuccessful, although the existence of an hysterical form of this disease must be admitted. In other cases the affection is closely related to epilepsy. Myoclonia cannot, however, be classed with epilepsy, even although, apart from Unverricht's type, myoclonic symptoms may be present between the attacks of true epilepsy (Reynolds, Ballet, Dide, Hoffmann, Bruns), and may be present for a considerable time before these attacks first appear. Of another group of cases, all we can say is, that the disease is due to mental degeneration. I do not think the attempt to classify this neurosis—even Unverricht's type of it—as a variety of chronic chorea (Möbius, Böttiger, F. Schultze) is justifiable, and Lundborg emphatically objects to it. Schupfer regards Unverricht's form alone as a disease *sui generis*, myoclonia otherwise being, in his opinion, merely a symptom of various diseases. Dana seems to hold a similar view.

The non-hysterical form of myoclonia is, in my opinion, an independent disease, the cases described by Unverricht forming a special type of it, or a disease of other character and origin. In addition there are some affections of an obscure nature which include myoclonic contractions among their symptoms. Finally, it must be admitted that the hysterical character of myoclonia is not always easy to recognise. The points mentioned as regards the diagnosis in the chapter on hysteria should be taken into consideration.

Henoch's chorea electrica is probably identical with myoclonia, but this name has been given to very different conditions (see section on chorea).

Lugaro and Soury (*Ann. méd.-psychol.*, 1897) and others regard myoclonia simply as a symptom, a manifestation of the "neuroclonic condition" of the motor neurones. According as the motor protoneurones, the subcortical neurones of the second order, or the psychomotor cortical neurones are affected, the resulting clinical symptoms will take the form (1) of fibrillary tremor and paramyoclonus, (2) of tic, or (3) of the *maladie des tics*. Raymond (*R. n.*, 1904) thinks that myoclonia may be caused by hysteria, psychasthenia, or epilepsy, and may also accompany organic diseases of the nervous system. Huchard-Fiessinger (*Rev. de Méd.*, 1905) are of opinion that the disease is not a simple entity and that it may develop from various causes. Murri, with whom Seppilli, Patella, Massalongo, Clark, and Prout agree, attributes all these conditions

[1] *M. f. P.*, xvi. [2] *Schmidts Jahrb.*, 1888 and 1893. [3] *Il Policlinico*, 1901.

to the cortex of the motor area. Hunt thinks the origin is not cortical, but spinal. Dana attempts to classify the various forms; he distinguishes five types, but he includes too much under the term myoclonia. See also Fischer, *Gaz. des hôp.*, 1903. F. Schultze suggests the term monoclonia for convulsive tic, and that of polyclonia for general tic.

Further knowledge is required, as Heldenbergh himself admits, before we can decide whether the functional intermittent paradoxical myoclonus which he describes, and the chief symptom of which is clonic contraction in the antagonists on the attempt to set a group of muscles into action, *e.g.* in sudden contraction of the extensors of the hand and fingers in the effort to grasp an object, can be considered as an independent disease.

The classification and origin of the " nystagmus-myoclonia " observed in Brittany by Lenoble-Aubinean (*R. n.*, 1906) is as yet obscure. It is a hereditary, familial disease, in which congenital nystagmus, tremor of the head, and fibrillary tremor are associated with myoclonic and other symptoms. The affection is apparently incurable but is not progressive. No pathological changes were found in the cases examined (*R. n.*, 1907).

One form of the disease has been distinguished in which the symptoms of motor irritation are said to be exactly similar to fibrillary tremor. Some authors (Dana) would therefore regard myokymia as a form of myoclonia. I think it very doubtful whether such affections should be included in this class, but it must be admitted that paramyoclonus has been described as a symptom of various diseases of the spinal cord—myelitis, poliomyelitis, etc.—by Raymond, Lévi-Follet (*R. n.*, 1900), Fargue, etc. I am convinced that these cases should not be regarded as myoclonia, but rather as instances of an unusually severe and extensive fibrillary tremor. It is certainly difficult to gather from the description of some cases whether there was merely a fibrillary tremor or an actual co-existence of two different diseases. Thus Feindel and Froussard (*R. n.*, 1899) describe the combination of myoclonia with multiple neurofibromatosis, Lévi and Follet with spondylose rhizomélique, etc.

In non-hysterical cases the *prognosis* is grave. Friedreich thought recovery had taken place in one case, but a relapse followed and the condition became chronic. In a few cases electricity had an excellent effect, but these were probably of an hysterical nature.

Careful examination of the nervous structures, which Schultze made in one case, gave an entirely *negative* result, but Hunt on the other hand found hypertrophy of the primary fibres of the muscles.

The Nissl changes in the pyramidal cells of the cortex described by Clark and Prout are hardly worth considering. Mott has found similar changes in the cells of the motor area in a case which was apparently of Unverricht's type.

Friedreich believed this neurosis to be due to a condition of irritation in the nerve-cells of the anterior horns of the spinal cord. Unverricht and Carrière are of the same opinion. Symptoms of the same kind have been observed in animals after removal of the thyroid gland (Wagner). Lundborg attributes it to auto-intoxication.

He seems inclined to think that the parathyroid glands have something to do with the causation (*Z. f. N.*, xxvii.). Valobra speaks of an infective form of myoclonia (*Morgagni*, 1904).

Treatment.—Bromide of potassium and chloral hydrate have a calming influence and may produce temporary improvement. Arsenic has done good in a few cases. The galvanic current is worth a trial, and static electricity and the increasing faradic current have also been recommended. Thyroidin is said to have been successful in one case. These drugs have all proved unsuccessful in my cases. Psychotherapy may sometimes be of service.

Occupation Spasms

(THE OCCUPATION NEUROSES ; CO-ORDINATED OCCUPATION NEUROSES)

Literature in Bernhardt, Nothnagel's "Handbuch," xi., second edition, 1904; and Remak, article on occupation neuroses in Eulenburg's "Realenzyklopädie," fourth edition.

By an occupation neurosis we understand a disturbance of the innervation of the muscles, which occurs only in certain complicated *movements which are acquired by practice*, the muscles responding to the will in every other action. This condition is specially described by Bell, Duchenne, and Benedikt.

Writers' Cramp (*graphospasm*, *mogigraphia*) is the most common form—the type of this affection. It is a disturbance of the innervation of the muscles used in writing, in consequence of which writing becomes difficult or impossible and the handwriting deformed and indistinct, although the hand may be used for all other purposes. The exciting *cause* of writers' cramp is *overstrain* of the muscles by writing. The condition usually develops in those whose occupation entails much writing, but it practically never affects persons with a normal nervous system. In the great majority of cases there is a *neuropathic* predisposition. Almost all my patients have been *neurasthenics*; some of them have suffered from *migraine*, others from *neuralgia*, one from *stuttering*, one from severe *vertigo*, another from *epilepsy*. Writers' cramp has also been combined with *convulsive tic*, *agoraphobia*, *tabes*, a previous *poliomyelitis*, *acrocyanosis*, etc. One of my patients had a congenital form of diplegia which clinically showed a great resemblance to disseminated sclerosis. The neuropathic origin of the condition is shown by the fact that it may affect *several members of one family*.

The use of hard-pointed steel pens, and in particular a wrong *method of writing*, *i.e.* one associated with an unnecessary degree of effort, help to bring on the condition. The method in which the little finger serves as a support for the hand, the muscles of the fingers alone being used in writing, is particularly bad. The greater the strain upon the small muscles of the hand, the more likely is it that the spasm will develop. In one lady the condition commenced after she had nursed her husband for paralysis agitans, a disease from which she was afraid she too might suffer. In another case the writers' cramp occurred directly after a spiritualistic séance. I might take this opportunity of mentioning that these incubators of mental infection have furnished me with a number of cases of psychoses and neuroses (a statement confirmed by Henneberg).

It is obvious that adult males will most frequently be affected, but even children are not immune.

A state of prolonged emotional strain may produce the condition. In only a few cases has it followed injuries to the hand or local inflammation (Seeligmüller). There can be no doubt, however, that it may be produced in a *reflex* way by such conditions of pain, possibly also by a neuritis.

The disorder develops gradually. The patient first becomes tired when he writes for any time; then he notices that he has not the same control over his pen as formerly, that he does not write so rapidly or smoothly, and sticks at a letter here and there. After a time he becomes aware that the muscles contract spasmodically, the pen-holder being gripped much too tightly.

In the majority of cases the difficulty is produced by this spasm. The *spastic* form of writers' cramp is the ordinary one. The tonic spasm chiefly affects the flexors of the fingers, notably those of the thumb and index-finger. In rare cases the extensors are spasmodically contracted, so that the pen is not grasped and falls from the fingers. The fingers are sometimes forcibly separated, the thumbs being drawn into the palm of the hand by the spasm. Gradually it extends to the muscles of the hand and fore-arm. The wrist-joint is over-extended, pronated, or supinated, the hand being thus lifted off the paper. I have seen other cases in which the spasm first attacked the abductors of the upper arm, and in this way the whole extremity was moved away from the table. As a rule, writing causes *pain* in the muscles, bones, or joints. At first the patient can continue writing for some time before the spasm becomes troublesome, but the difficulty gradually increases until he is unable to write a single word without showing the effect of the spasm. The written characters become *rude*, *irregular*, *interrupted* and *broken*; some of the strokes are *incomplete*, the down-strokes too thick, some letters too small or too large. If we observe the patient writing, we see that his hand is to a certain extent stiffened upon the penholder, the pen pushed up between the index and middle fingers, or the hand forcibly lifted from the paper. The more he

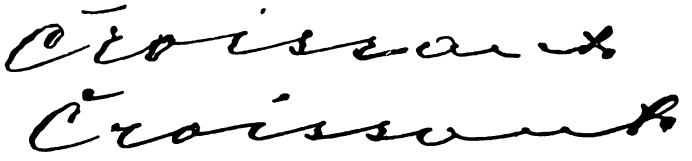


FIG. 415.—(1) Written under the physician's observation. (2) Written alone.

thinks about it, and the more he is annoyed by the condition, the worse does it become (see Fig. 415).

Writing is less frequently disturbed by *tremor*; this is known as the tremulous form of writers' cramp. In other uncommon cases simple *weakness* becomes evident in writing. The hand, which has otherwise normal muscular force, gives way in writing, and the muscles become paralysed (*paralytic* form). A combination of spasm and tremor is not uncommon.

Finally, we have a *sensory* or *neuralgic* form of this neurosis. A painful feeling of fatigue may, indeed, be felt in any of the forms just described, especially the spastic, but there are cases of writers' cramp in which pain alone makes writing impossible and is felt only during the action of writing.

Objective examination as a rule yields a *negative* result. Motility and sensibility are unimpaired, and there is neither ataxia nor tremor. Pressure-points on the nerves are also rarely observed. Occasionally swelling and thickening on the tendon-sheaths of the extensors of the fingers (A. Pick), or periostitis on the external condyle of the humerus (Runge) have been described, but I think there is no doubt that these only produce writers' cramp in predisposed individuals. The presence of *neurasthenia* may be revealed by an increase of the tendon reflexes, exaggeration of the mechanical excitability of the muscles, vasomotor disturbances, etc.

The *course* of the disorder is almost always protracted ; it may persist for years or as long as the patient lives. As a rule he tries to learn to write with the left hand. This may succeed for a time, but the left hand usually becomes affected sooner or later.

The *prognosis* is not very favourable. Recovery is the exception, and in cases of apparent recovery *relapses* are frequent. The longer the duration of the trouble, the less prospect is there of a cure. I have seen complete recovery in a few cases, and so have Berger and others ; in one case the patient, a young girl, recovered when she became betrothed, but *relapsed* after her marriage. The prognosis is better in the sensory form. Zabludowski's experience has led him recently to speak very favourably of the prognosis in this form.

The *diagnosis* is usually an easy matter. One should beware of regarding every disease which is characterised by a disorder of writing as writers' cramp. Disseminated sclerosis, tabes (commencing in the upper extremities), a slowly developing right hemiplegia, paralysis agitans, and other organic diseases of the nervous system may give rise to disturbances of motility, sensibility, and co-ordination which are at first noticed by the patient only in complicated movements, such as writing. The physician, however, will find on close examination that this symptom is apparent in simple testing of the motility, sensation, co-ordination, etc. I have known a few cases in which agraphia was wrongly diagnosed as writers' cramp. The diagnosis is difficult only in cases where the cause of inability to write is *hysterical* or *neurasthenic* tremor. The fact that other symptoms of hysteria and neurasthenia are present does not exclude the possibility of writers' cramp, as it is frequently associated with these neuroses. If, however, it is merely a *consequence* of the nervous tremor, the prognosis is very much more hopeful than if it is an independent affection. The diagnosis can as a rule be easily inferred from the fact that nervous tremor becomes evident in the same way in all the other movements, and the cramp in cases of this kind can generally be greatly diminished by distracting the attention or by the effect of suggestion. These signs are not always present, nor always to be depended upon. It is also the case that in severe, long-standing writers' cramp the symptoms—spasm, tremor, or weakness—may appear in other fine manipulations of the hand, such as threading a needle, sewing, violin-playing, etc.

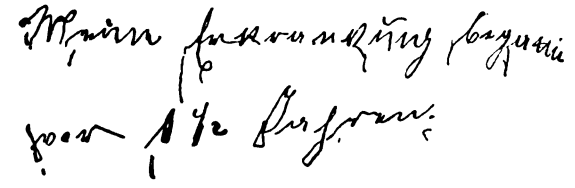
Pathological changes have not hitherto been found, nor are they likely to be found in the future. The disease is undoubtedly a *purely functional* one—an *exhaustion neurosis* which has its seat in the co-ordinating centres, the central system which controls the associative actions of the muscles which are used in writing. The centrifugal and sensory excitations which accompany writing, and which are not normally perceived, become conscious and create a feeling of discomfort ; the intensity of the cramp therefore corresponds to the amount of attention which is directed to the act of writing.

Treatment.—The quickest remedy is *absolute avoidance of the act* which brings on the spasm. Writing should therefore be given up for a long time. It should also be ascertained whether a faulty method of writing and guiding the pen is the cause, and if so a *change in the method of writing* should be recommended. Teachers can greatly aid in preventing writers' cramp by seeing that the child from the first acquires the habit of writing in the easiest possible way. The use of thick penholders or of a cork

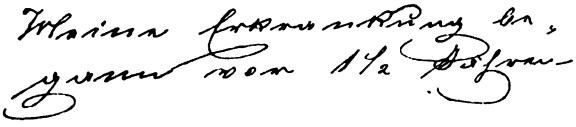
slipped on to the holder, may facilitate writing, and the child should be taught to form large letters in round Latin characters.

Zabludowski [1] has collected the various recommendations as to the attitude and position of the hand and fingers in writing, etc. He advises chiefly practice at first in writing large round capital letters, in which the hand is moved freely over the paper, a transition being gradually made from this to the ordinary handwriting. He recommends that after two or three words have been written the forearm, held in the parallel direction, should be moved from left to right ; that at the places where the pen is apt to stick, a rest should be taken, etc. etc. Gowers advises that writing should be done from the shoulder only, whilst Böttiger has seen the best results from exercise of the muscles of the thumb, index, and middle fingers. A pencil or quill pen is preferable to a steel pen. Penholders may be had with a kind of ring which may be passed over the finger. In some cases *Nussbaum's bracelet*, which holds the spread-out fingers together, is of service, as the following illustration shows (Fig. 416, *a* and *b*).

416 *a*.



416 *b*.



FIGS. 416*a* and 416*b*.—Test-writing of a patient with writers' cramp, without and with the Nussbaum bracelet. (After Limbeck.)

Appliances of similar kinds and special penholders have been recommended by Zabludowski. Close study of the symptoms in each individual case will suggest measures whereby the abnormal muscular tension and the anomalies of position which they cause may be counteracted. Thus, when Limbeck found his bracelet to be no longer successful, he provided it with a splint, which prevented the fingers assuming the extreme position of flexion. If local changes, such as swelling of the tendon-sheaths, are present, treatment must naturally be directed to these.

Drugs have little effect upon the disease, although sedatives and nerve-tonics (*e.g.* strychnine) have been recommended, and recently treatment by opium and bromide (Dornblüth), the indications for which do not seem to me to be satisfactory.

Hydropathy may be of great service, especially if the cramp is due to neurasthenia. A stay at the seaside, in the hills, etc., may also be good. *Electricity* is very uncertain in its effect. The galvanic current is used and is passed through the cervical cord, or the cathode is placed in the nape of the neck and the anode in the region of the brachial plexus and the various nerves of the arm. M. Meyer recommends the stabile anode treatment of possible pressure-points on the spinal column.

[1] *N. C.*, 1903 ; *Z. f. physik. und diät. Therap.*, vii.

The best results are obtained by *massage* and *gymnastic exercises*. Swedish gymnastics (movements of the various muscles of the hand and fingers against resistance) have proved most serviceable in my cases. Further details are given by Zabludowski and Konindjy,[1] but the theoretical considerations of the latter seem to me very much open to dispute. Bum is in favour of Zander and Herz's apparatus. Hartenberg [2] recommends the application of elastic bands for twenty minutes twice a day. There is no doubt that suggestion plays the chief part in the treatment in many cases.

When every remedy fails, the patient should be advised to use a typewriter.

Other Occupation Neuroses

These neuroses are almost as numerous and varied as the kinds of occupation. Cramp of the piano-player, the violinist, the flutist, the 'cellist, the needlewoman, the telegraphist, the tailor, the cobbler, the cigar-roller, the watch-maker, the milker, the blacksmith, and the dancer have been described, and many other forms as well.

The description given of the development and symptoms of writers' cramp applies also to these forms.

Piano-players' cramp is specially apt to occur during training. As a rule it consists in abnormal muscular contractions, one or more fingers being thus lifted off or pressed down upon the keys, so that the playing is interrupted. This form of cramp is usually painful, and the pain is felt not only in the fingers and arms, but specially in the shoulders and spinal column. According to Zabludowski, the trouble is often caused by traumatic arthritis. There is also a paralytic form of piano-players' cramp, the hand or some fingers being completely paralysed. In most cases the pain and fatigue occur only during playing. Violinists' cramp may affect the bowing hand, the string hand, or both. In one case under my care paræsthesiæ appeared on every attempt to play the violin, which had therefore to be discontinued. As soon as the patient ceased playing, the symptoms entirely disappeared. Another patient had pain in the muscles of the shoulder and upper arm. Flute-players' cramp affects several fingers or the whole hand, the left more often than the right.

Blacksmiths' cramp attacks the muscles of the upper arm and shoulder, especially the deltoid and biceps, which become tonically contracted when the hammer is lifted. The pain associated with the spasm is intense.

Milkers' cramp occurs in dairymaids who have daily to milk cows. Milking is accompanied by tonic spasm in the flexors and extensors of the hand and fingers, accompanied by severe pain. The cramp is sometimes associated with a feeling of numbness and cold in the fingers. Remak describes a combination of this spasm with degenerative neuritis in the median nerve. By the name of *waiters' paralysis*, Runge has described weakness of the extensors and supinators of the hand, due to the carrying of many plates, glasses, etc., entailed by the occupation of waiting.

The cramp of seamstresses or tailors affects chiefly the muscles of the thumb and index finger, that of cigar-rollers attacks the hands during the rolling of cigars. In telegraph-operators the spasm becomes evident in working the dots and lines of the Morse system. Working with the Hughes apparatus may also produce the neurosis, as the cases of Cronbach show (*A. f. P.*, xxxvii.).

I have twice seen a *keirospasm* or *xyrospasm*, *i.e.* painful contraction of the muscles of the hand and fingers which occurred in a barber every time he attempted to shave. This should be distinguished from the *keirophobia* which I have also observed, and which is a condition of anxiety which overcomes the barber just as he is on the point of shaving a customer.[3] An occupation neurosis may also appear in diamond-cutters (Stephan).

The professional paresis of leather-dressers (tawers) mentioned on p. 548 (Höflmayer) seems also to be an occupation neurosis. So-called tennis-arm (Clado, *Prog. méd.*, 1902) is probably of the same nature.

The sawyers' cramp described by Poore is apparently an occupation paresis. I have observed

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[1] *Z. f. diät. Therap.*, viii.; *Nouv. Icon.*, xviii. [2] *Arch. de Neurol.*, 1906.

[3] The condition of anxiety and vertigo is of course much more apt to occur in the person who is actually being shaved than in the barber.

an occupation spasm in the muscles of the lips in a trumpeter; whenever he put the instrument to his mouth a spasm occurred in the orbicularis oris, and he was unable to produce a note. It was impossible to ascertain whether the condition was a spastic or a paralytic one. Strümpell and Stadler (*M. m. W.*, 1903) have reported similar cases.

Stammering is closely allied to occupation spasm. B. Fränkel (*D. m. W.*, 1887) describes painful fatigue of the muscles of the vocal cords in professional speakers, which he calls mogiphonia.

Occupation spasms less often affect the lower extremities, but a dancers' cramp, consisting of tonic, painful contraction of the calf muscles, which sometimes occurred when the dancer glided forward on tip-toe in beginning to dance, has been described. A cramp may also appear in persons who have to work a treadle. See also the paper by Stürtz (*Charité-Annalen*, 1903).

Occupation neuroses may affect the *eyes*. Thus in constant use of the microscope, *e.g.* by meat-inspectors, a spasm may develop in the muscle of accommodation. Orbicularis spasm has also been observed in watchmakers (T. Cohn), and there is a watchmakers' cramp in the hands (Wilde). Tranjen (*B. k. W.*, 1892) describes spasm of the ocular muscles in soldiers developing during military drill with the eyes fixed. "Miners' nystagmus" may also be included in this class. I have found this condition in a violinist who had to read music from a high stand under a bad light.

In the *diagnosis*, care should be taken not to mistake these occupation neuroses for occupation paresis (professional paresis, see p. 548), *i.e.* the paralytic conditions which develop in certain muscles as a result of their over-strain. This paralysis is usually due to neuritis. Careful examination prevents this mistake being made, as the paralysis is usually chronic and degenerative and often associated with sensory disorders. The two conditions have been confused by some writers, *e.g.* Savill. The occupation neuritis may of course be combined with the neurosis. I have, for example, observed this combination in a few instances in violinists and 'cellists.

Treatment is practically the same as that described under writers' cramp.

For pianists, Zabludowski (with whom Bum agrees) recommends the use of a special piano, in which the dimensions are considerably smaller than in the ordinary piano, with a gradual transition from the use of the small to the large keys.

Tetany

(Tetanille, Idiopathic Muscular Spasms, etc.)

Bibliography in Frankl-Hochwart, "Die Tetanie," Nothnagel's "Handbuch," second edition, 1907.

This disease, first described by Steinheim and Dance, consists in *intermittent, bilateral, tonic*, and usually painful spasms, occurring chiefly in *certain groups of muscles* of the upper extremities, unaccompanied, as a rule, by any impairment of consciousness.

In some districts and countries, such as Sweden, this is a rare disease, whilst in others, Vienna for example, it is *epidemic* during certain months (March and April). Young men of from sixteen to twenty-five years of age are peculiarly liable to it, the patients belonging almost entirely to the *working* classes. It is not uncommon in childhood. Women are hardly ever affected except during *pregnancy* and *lactation*. Old age is almost immune.

A number of facts seem to show that tetany has a *toxic-infective* origin: 1. its *epidemic* and *endemic* occurrence, and its tendency to affect several members of a family; 2. its development in association with *infective diseases*, in the prodromal stage, in the course of, and after typhoid, cholera, less often scarlet fever, measles, pneumonia, influenza, tonsillitis (J. Grünewald), etc.; 3. the part played by some *poisons* (ergotin, alcohol, chloroform, lead, phosphorus, extract of male fern—I have even seen it develop directly after a spermin injection with symptoms of

collapse, and, in one case, after eating lobsters); the tetany of nephritis should possibly be included; 4. the connection between tetany and *gastro-intestinal disorders*, to be discussed later; and 5. the toxic products found in the stomach (Bouveret and Devic, Amato) and in the urine of patients with tetany by Ewald and Albu. The value of these observations has been disputed by Fleiner, Sievers, and others.

Other facts cannot without further evidence be taken as supporting the view that a virus is concerned; indeed, they seem to show that tetany must have some other cause. Chief among these is the remarkable fact that certain *occupations* create a marked predisposition. Thus among three hundred and fourteen male patients (Frankl-Hochwart) there were a hundred and forty-one shoemakers and forty-two tailors; or, according to his latest statistics, among five hundred and twenty-eight cases there were two hundred and twenty-three shoemakers and one hundred and seventeen tailors; whilst other trades, building, weaving, etc., only furnished one or two cases. This tendency of tetany to attack shoemakers had struck the earlier writers, and led to its being regarded as a "cobblers' cramp," or an occupation neurosis. I think, however, that the occupation is not a direct cause, but that by bringing the patient into connection with some unknown toxin—possibly adhering to the leather which he uses—it acts as a predisposing cause. According to Voss, metal-workers are chiefly affected in St Petersburg.

Another observation which may be brought into harmony with the toxic theory is that tetany very often follows *total removal of the thyroid gland* (Weiss-Billroth, Eiselsberg), whilst partial extirpation hardly ever has this effect. In two of the cases where tetany followed partial removal of the gland, the patient was pregnant (Meinert, Vassale). Some writers regard enucleation of the parathyroid glands as a cause (Vassale, Jeandelize, etc.). I shall return later to this subject. Lundborg has specially drawn attention to the individual differences in the results of thyroidectomy.

Healthy children seldom suffer from tetany, which is comparatively common in those who have *diarrhœa* or rickets (Ganghofner, Kirchgässer, Hochsinger), but Kassowitz goes much too far when he regards these conditions as a cause of tetany. Tetany in children may be combined with laryngismus stridulus (Loos, Escherich) and general convulsions. So-called latent tetany may also undoubtedly cause laryngismus; on the other hand Moos's theory that spasm of the glottis is always a symptom of tetany cannot be accepted (Hauser, Bendix, Brandenburg). Ganghofner and Japha think this spasm is usually caused by tetany. Our knowledge of the tetany of children and its relation to rickets and laryngismus is still very indefinite (see further on). Tetany may be associated with osteomalacia (Blaziczek, Freund).

Adults who suffer from gastro-intestinal affections are sometimes affected. A specially severe form has been observed in *gastric dilatation* (Kussmaul). It has not so far been definitely decided whether the toxic agent is the cause of the spasm or whether the spasm has a reflex origin. The fact that it has occasionally occurred directly after the use of a *stomach-pump* or the passing of a tube, or after profuse vomiting, seems to point to a reflex origin, and the observation—which cannot be implicitly relied upon—that the spasm has disappeared after the evacuation of entozoa confirms this theory. It is also remarkable that Gerhardt was able in a

case of tetany with dilatation of the stomach to bring on the spasm by slight percussion of the stomach. Kussmaul, Fleiner, and others think that diminution in the amount of fluid in the body and thickening of the blood is the cause of the attacks. Fleiner has proved this directly, but his theory is opposed to that of auto-intoxication, which is more and more gaining ground.

Chill, exhaustion, and emotional excitement are also thought to be exciting causes. During recent years the theory of the *parathyroid origin of tetany* has gained in favour. It is specially advocated by Vassale-Gennari, Eiselsberg, Kocher, Pineles, Erdheim, Lundborg, Chvostek, and Loewenthal.

These writers describe the parathyroids as a pair of glands, one upper and one lower, about 6 to at the most 15 mm. in size, of a brown or yellowish colour. These are situated in front of the posterior surface of the lateral lobe of the thyroid, at the point where the two branches of the

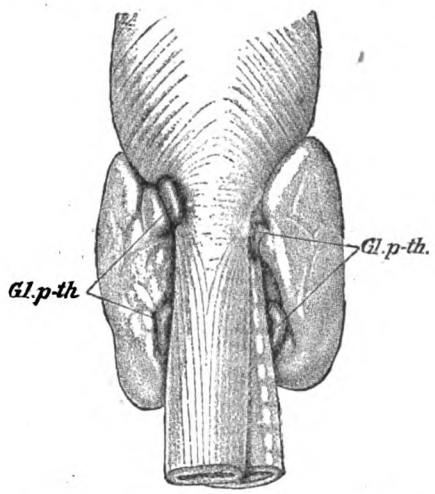

FIG. 417.—The thyroid gland with parathyroids (gl. p. th.) in man, after Zuckerkandl. (Seen from behind.)

inferior thyroid artery enter the gland, in the neighbourhood of the recurrent (see Fig. 417). They were first described by Sandström. Details as to their anatomy and histology will be found in the papers by Peters (*V. A.*, Bd. clxxiv.), Richardson ("The Thyroid and Parathyroid Glands," Phila., 1905), Alquier (*Gaz. des hôp.*, 1906), and in the exhaustive review by Schirmer (*C. f. Gr.*, 1907).

All the published cases of thyroidectomy and the experiments of Gley, Biedl, Pineles, and Erdheim show that tetany is caused by removal of the parathyroid glands or the so-called epithelial corpuscles. Some of these writers go so far as to attribute tetany generally to absence or insufficiency of the parathyroids, which they regard as organs for destruction of poisons; the other factors they consider merely secondary or exciting causes. Thus the poison in dilatation of the stomach would only cause tetany if the epithelial corpuscles were insufficient; the professional over-strain of the muscles in cobblers would only give rise to the spasms if the parathyroids were congenitally inferior, etc. This theory makes it very difficult to explain the tetany of children. Pineles and Erdheim believe in this

parathyroid origin, but Heubner,[1] Finkelstein,[2] Soltmann, and Kassowitz[3] do not. They point out that the tetany of children is distinguished clinically by the tendency to convulsions, spasm of the larynx, and chronic muscular contractions, and for this reason they have given it the name of *spasmophilia*. Stoeltzner[4] thinks it is due to calcium poisoning from cows' milk, and Quest and R. Weigert[5] attribute it to want of chalk in the organism. In any case the cause is not definitely understood, and the view of Jacobi,[6] that both internal and external poisons, *i.e.* those originating in, and those introduced into the organism, may produce the disease, is probably nearest the truth.

Symptomatology.—The tonic muscular spasms of tetany do not as a rule come on suddenly and unexpectedly. There are usually *prodromata* in the form of paræsthesiæ and pain in the limbs, less often of disturbances of the general condition, such as headache, vertigo, and a feeling of exhaustion.

The upper extremities, especially the muscles of the hand and fingers, are chiefly and often solely affected. In typical cases the *interossei* and the other small hand muscles are so markedly involved that the position of the hand and fingers is quite characteristic and the disease can often be recognised at a glance. The basal phalanges are strongly flexed, the middle and terminal phalanges extended (Fig. 418), the fingers pressed together, and the thumbs turned inwards until they are covered by the other fingers. The hand itself is usually flexed and the forearm often flexed on the upper arm. In one case the fingers were so tightly pressed together that dry gangrene developed. The hands do not always assume this *writers'* or *accoucheurs' position*. The fingers may be flexed, the hand clenched, or the fingers spread out and extended at every joint. The cramp is very seldom confined to single fingers.

The *lower extremities* are often involved, particularly the flexors of the foot and toes, which become tonically contracted, the toes being turned into the sole, the foot curved into a hollow and assuming the equino-varus position. All the muscles of the legs may be affected.

The muscles of the *trunk*, *tongue*, *pharynx*, *larynx*, and those of *deglutition* and *respiration* are only involved in severe cases. The ocular muscles may also be affected (Kunn), and tonic contractions in them may cause strabismus. Even the sphincter pupillæ and ciliary muscles may be involved.

The attack of spasm may last only for a few minutes, for several hours, or even for as long as ten days. It is sometimes so mild that the muscular contraction can be overcome without a special effort; at other times, and always in severe cases, it may be so intense that the physician cannot change the position of the limb from that into which it is forced by the spasm, and any attempt to do so causes great pain. The muscular contractions are usually in themselves very painful. The attack gradually passes off, but a sensation of tension and other paræsthesiæ may continue after it is over. It is much rarer for the muscles to remain paretic for some time after the spasm has left them, but a certain muscular weakness is often present in the intervals (see further on).

The mind is not affected during the attack; mental confusion has been

[1] "Lehrb. d. Kind.," i.
[2] "Lehrb. der Säuglingskr.," Berlin, 1905.
[3] *W. kl. W.*, 1906.
[4] *Jahrb. f. Kind.*, Bd. lxiii. and lxiv.
[5] *M. f. Kind.*, 1906.
[6] *Z. f. N.*, xxxii.

observed only in rare cases, and almost exclusively in the toxic forms of tetany, but it is not uncommon in the tetany of children, which is sometimes associated with eclampsia or epilepsy.

The *temperature* is slightly raised in many cases and may reach 38·5 to 39° C. (101 to 102·2° F.). A subnormal temperature has also been frequently recorded. The pulse is usually rapid during the attacks.

The spasms may be separated by intervals of hours, days, or weeks;

FIG. 418.—A girl with tetany during the spasm. (Oppenheim.)

as a rule they follow in quick succession, and in some cases the muscles are never completely relaxed. They are sometimes brought on by active movements, mechanical irritations, etc.

The spasms are the most important symptom of the disease, but there are a number of other characteristic signs. These include—

1. *Trousseau's Sign.*

Trousseau found that an attack could be provoked by pressure upon the affected limb, " the nerve-trunks or the vessels being compressed in

such a way as to prevent arterial or venous circulation." In the great majority of cases deep pressure upon the internal bicipital groove during an interval between the spasms will bring on an attack. Sometimes it follows half a minute, sometimes only three to four minutes after the pressure is exerted. The anæmia caused by compression of the arteries was thought to be the cause, but this is disproved by the fact that in some cases the attacks can be brought on from other points at which there are no main arteries, *e.g.* the wrist-joint. Frankl-Hochwart then showed that the attack is caused by compression of the nerves—probably of a reflex nature. Trousseau's sign is not found in any other disease of the nervous system, but its disappearance is not always a proof that the condition is cured.

2. *Increase of the mechanical excitability of the motor nerves* (Chvostek's symptom).

Slight percussion of the motor and mixed nerves may produce lively contractions in the muscles which they innervate. This is most apparent in the *facial* muscles, and is shown by the so-called *face-phenomenon*, viz., marked twitching in the muscles of the face produced by passing the handle of the percussion-hammer or the tip of the finger over the pes anserin. major. Increase of the mechanical excitability of the nerves is present in other diseases of the nervous system, in tuberculosis, simple dilatation of the stomach (Heim), and enteroptosis (Mager), but seldom to such a degree as in tetany. On the other hand this sign is usually not very marked in the tetany of children (Schlesinger), but some writers (Escherich, Thiemich) describe a lip-phenomenon—a snout-like protrusion of the lips on percussion of the muscles of the mouth—as occurring here. The investigations of Toulouse and Vurpas show that this symptom is not peculiar to tetany, but occurs in various other affections.

3. *Increase of the electrical excitability of the motor nerves* (Erb's symptom).

This sign is present in almost every case, as shown by the fact that a very weak current will be produced by the CCC, that a very slight increase of the strength of the current will produce CCTe (thus the ulnar nerve reacted to 0·1 M.A. with CCC, and to 0·5 with CCTe), and that AOTe and sometimes even COTe may be produced.

In one case of very severe tetany which I examined, galvanic stimulation was always immediately followed by CCTe; this was produced even by the weakest current, and a simple CCC could not be maintained.

According to the investigations of Mann and Thiemich, the COC may be obtained by weak currents, and they regard this as a very characteristic symptom.

Apart from the first weeks or first two months of the child's life, the nerves do not readily respond to electricity in early childhood. Mann found the average current, *e.g.* for the median nerve, to be: faradic, 110·4 Ra; galvanic, CCC 1·41 M.A., ACC 2·24, AOC 3·63, COC 8·22; in tetany on the other hand the average was: CCC 0·63, ACC 1·1, AOC 0·55, COC 1·94. If the COC is produced under 5 M.A., tetany is certainly present.

According to the investigations of Pirquet (*W. m. W.*, 1907), the appearance of an AOC below 5 M.A. is a sign of pathological increase of excitability.

Increase of the excitability for the faradic current is less constant.

4. *Increase of the mechanical and electrical excitability of the sensory nerves* (Hoffmann's symptom).

This is present in many cases. Slight pressure on the supraorbital, ulnar, great auricular, long saphenous, and other sensory nerves may produce a sensation described as radiating, viz., paræsthesiæ in the region innervated by the nerve. Similar pressure in normal persons would only produce a local sensation, if any. The normal feeling caused by a blow or hard pressure upon the ulnar nerve at the elbow-joint is in tetany produced by weak stimuli. This symptom is not, however, pathognomic for tetany, as I have often found it in neuropathic individuals. We know little regarding the increase of the electrical excitability of the sensory nerves.

In healthy persons weak galvanic currents at first produce a CSe (a sensation of cathodal closure), which soon develops into a persistent CCCS sensation of current followed by ASe and ACCS. Finally, the increase of the strength of the current produces not only local but also radiating sensations. In tetany these are brought on by much weaker currents, and the intervals between the local and the radiating sensations are so short that they may almost follow stimuli of the same strength.

Increase of the electrical excitability has also been found in the *auditory* nerve (Chvostek junior).

Bechterew points out that repeated stimulation may produce a progressive increase of the mechanical and electrical excitability. He calls this the reaction of excitation. Kashida (*Mitt. d. med. Fak.*, Tokio, v.) has shown that there is also increased excitability of the motor nerves to thermal stimuli. P. Philippson (*B. k. W.*, 1907) found that the electrical excitability might be diminished if the galvanic current were continuously passed through the nerves for a considerable time.

Inconstant Symptoms.—Changes in secretion have been observed in tetany, including hyperidrosis (especially after the attacks), and much less often polyuria, albuminuria, and glycosuria. Redness, œdema of the skin, urticaria, herpes zoster, and abnormal pigmentation have been noted. *Loss of the hair and nails* is an interesting but uncommon symptom.

Paralysis in certain muscles—especially those of the sacral, pelvic, and lumbar regions—with consequent disturbance of gait (waddling gait), has been observed in a few cases (Hoffmann, Weiss, Kalischer, Fuchs). Atrophy and anæsthesia are quite uncommon.

The *reflexes* are normal, exaggerated, or diminished, and Westphal's sign has occasionally been found. *Mydriasis, rigidity of the pupils, optic neuritis* (?), *cataract* (reported by Magnus, Peters, Bernhardt, Freund, Saska, A. Westphal, and Wettendorfer) are very rare symptoms. Uhthoff found a condition between tetany and myotonia in a patient with cataract. Westphal's sign and immobility of the pupils were present in a case of tetany due to thyroidectomy. In another similar case Hoffmann found the myotonic reaction which has been occasionally noted by other writers. In one very complicated case Freund saw myosis of the corresponding pupil during the attack which was artificially produced (Trousseau). *Mental* disturbances in the form of hallucinatory confusion have occasionally been described (Frankl-Hochwart). Schultze,[1] Voss, and recently Lapinsky,[2] have made similar observations. Tetany, especially the form following removal of the thyroid, is not infrequently associated

[1] *B. k. W.*, 1897.

[2] *N. C.*, 1907.

with epileptic conditions (Krönlein, Kraepelin, Hahn, Ehrhardt). Freund[1] has seen an attack of tetany pass into a condition of profound unconsciousness with loss of the pupil reflex, etc.

Tetany may also be combined with myxœdema, exophthalmic goitre, and other affections.

Fraisseix deals in his thesis with the combination of tetany and exophthalmic goitre. The co-existence of exophthalmic goitre, sclerodermia, and tetany is described by Dupré-Guillain. A case mentioned by Brissaud-Londe (*Rev. de Méd.*, 1901), in which spasms of the type of tetany were in a gouty patient combined with other symptoms, is not quite clear.

Diagnosis.—The spasms are so characteristic that the disease can be easily recognised. Tonic muscular contractions of a similar kind certainly occur in hysteria and hysterical pseudo-tetany, but they are usually unilateral, and the Trousseau and Erb signs are absent, especially the latter. Hysteria and tetany may be combined, and general convulsions of an hysterical character may occur in the course of tetany. Freund has discussed in detail the relations of tetany to hysteria and epilepsy, and interesting cases of their combination have been published by H. Curschmann[2] and A. Westphal.[3] The attempt of some French writers to identify tetany with hysteria has not been successful, and we cannot accept the very similar theory of H. Curschmann. If the tetany involves the muscles of the trunk and jaw and those of respiration, it may have a superficial resemblance to tetanus, but the development of the spasms, their commencement in the muscles of the hand, and the late appearance of the trismus are important indications of the difference, and finally the intermittent character of the attacks of tetany as contrasted with the more continuous spasm of tetanus, etc., should prevent confusion.

I have seen an attack exactly resembling tetany develop in a woman with neurasthenic tachypnœa and vasomotor neurosis, after I had allowed her to run a few times through the room, but the persistent signs of tetany were absent.

It should be remembered that there is a form of *tetany without spasm*. The patient complains of paræsthesiæ, especially in the hands, and the symptoms of Chvostek and Erb are present. These *tetanoid* conditions may develop into true tetany.

Latent tetany occurs chiefly in childhood (Hauser, Escherich). According to Escherich,[4] the tetany of rickets is usually latent. Some authors regard laryngospasm, tetany, and eclampsia as various stages or manifestations of the same disease (Cheadle, Thiemich, Ganghofner, Japha, Finkelstein, etc.).

Tonic spasms, affecting more or less all the muscles of the body and accompanied by *albuminuria*, have occasionally been observed (Kussmaul, Kast). Their nature has not so far been explained. Kjelberg and Escherich have seen tonic spasm in children, lasting for weeks and months, and ending in recovery. This condition has a superficial resemblance to tetanus (pseudo-tetanus). In contrast to ordinary tetany, the muscles of the trunk are mainly affected, the arms are comparatively free, and the signs of Trousseau and Erb are absent. The prognosis of this condition is apparently favourable. Catanea and Guinon have each described a case

[1] *A. f. kl. M.*, Bd lxxvi. [2] *Z. f. N.*, xxvii. [3] *Charité-Annalen*, xxiii., and *B. k. W.*, 1907.
[4] *W. kl. W.*, 1890; *B. k. W.*, 1896; *M. m. W.*, 1907.

of this kind. Henoch reports similar conditions under the name of "*idiopathic contractures*," which chiefly affect the flexors of the fingers and toes. He has observed these in rickets, dyspepsia, eclampsia, etc., and is not inclined to consider them identical with tetany This is also the case as regards the forms of chronic flexion-contracture described under the name of "arthrogryphosis" by Rilliet, Barthez, Trousseau, Koppe, and others. In any case it seems to be desirable (as I have already said in the second edition) that experienced observers should make a sharp distinction between all these affections.

In the meantime this attempt has been made by Hochsinger.[1] Czerny and Moser had already described tonic contractions of the muscles or general muscular rigidity in infants with gastro-intestinal affections, and Hochsinger subsequently pointed out that the tendency to flexion-spasms in the extremities is very marked in infancy, and is indeed to a certain extent physiological during the first few weeks of life. Unfortunately he chose for this condition the name of *myotonia*, which was already associated with a definite form of disease. He regards arthrogryphosis (prolonged flexion-spasms in the upper and lower extremities, with the "fist-phenomenon" caused by pressure on the internal bicipital sulcus) and pseudo-tetanus as pathological degrees of this myotonia—conditions which should be distinguished from tetany. These conditions of contracture are permanent, painless, and not associated with changes of the mechanical and electrical excitability. The affection is caused by disturbances of the gastro-intestinal functions, skin diseases, and hereditary syphilis. He is inclined to ascribe them to pathological (?) changes in the anterior roots of infants, which Zappert has shown by the Marchi method. Kirchgässer and others have rightly objected to this view. Gregor was able to show that the general muscular rigidity and the galvanic hyper-excitability of the nerves in infants are both a result of artificial feeding, and may be cured by putting the child on mother's milk. This is shown by the investigations of Finkelstein and confirmed by Thiemich, Stoeltzner, etc. According to Gregor these conditions differ from tetany in their gradual development, their general extension, and their duration for months. In order to demonstrate the stiffness of the muscles, the child, when lying on his back, may be lifted up by the heels in one rigid piece. As we have already said, Heubner, Finkelstein, and others would distinguish the spasmophilia of children from tetany. But we must admit that many of the facts are still unexplained, and that the conditions of tonic spasm in infants require to be studied and classified.

The myotonic symptoms occasionally observed in tetany (Hoffman, Schultze,[2] Voss) are quite distinct from myotonia congenita.

Congenital contracture in one or more joints may, apparently, be caused by mechanical intra-uterine conditions, *e.g.* compression of the fœtus in the uterus by an abnormally scanty amount of liquor amnii and amniotic adhesions (Schanz, Weissenburg, Schiffer-Kalischer). These may gradually disappear.

The form of tonic muscular spasms occurring in rare cases of chronic lead-poisoning may differ from typical tetany (Haenel). Steinert (*M. m. W.*, 1905) reports one such case.

It is doubtful whether there is a form of tetany limited to single

[1] "Die Myotonie der Säuglinge," Wien, 1900, and "Die deutsche Klinik," etc., vii.
[2] *Z. f. N.*, xxvi.

muscles (*e.g.* tetany of the diaphragm with symptoms of asthma). Neusser has published a case of this kind.

Pathological Anatomy.—The investigations published so far (Tonnelé, Schultze, Berger, Kohts, Weiss, Schlesinger, Pick,[1] Ferranini, Rossolimo, Blum, Traina, Peters,[2] Becker,[3] etc.) have not led to any unanimous result. No changes were found in a few cases; in others there were changes in the spinal cord, partly hyperæmia and circumscribed hæmorrhages, partly cloudy swelling of the ganglion cells, pachymeningitis, softening, and an affection thought to be poliomyelitis. Changes in the brain, *e.g.* calcification of the vessels, have also been described. Although many of these changes indicate that the anterior grey matter of the cord is the starting-point of the disease, the results are as yet so few and contradictory as to be of little value.

Positive changes in the epithelial corpuscles have been reported by Erdheim and M'Callum.[4] Escherich [5] found numerous hæmorrhages in the epithelial bodies in the tetany of children.

Course and Prognosis.—The disease may run its course in a few days, with one or more attacks. It often lasts for weeks and months. There are also acute relapsing (Jaksch) and chronic forms, which, including remissions, may last for years. A duration of twenty years has been observed. Some persons get tetany every winter. It may also recur with each pregnancy (Hödlmoser).

The *prognosis* in regard to life is on the whole good, unless the patient is suffering from *gastric dilatation* (even tonic spasms of a benign nature may occur, according to Ury), or unless a *goitre has been removed* and myxœdema has developed. Even under these conditions the disease may be cured. Infants and children not infrequently die from the primary disease (intestinal catarrh), whilst otherwise healthy children as a rule recover. Tetany may prove fatal if it involves the respiratory muscles, in particular those of the diaphragm. In healthy adults the disease usually lasts for some weeks or months, but the careful statistics of Frankl-Hochwart [6] show that recovery is very often incomplete, a chronic tetany or invalidism being often the result. These statistics of course bear upon the tetany of adults, and of working people in particular. Cases following infective diseases and conditions of poisoning are characterised by a quick, mild course. I have seen such cases consist of a single short attack. Voss mentions having seen tetany recover during erysipelas, and in another case acute articular rheumatism apparently had the same curative effect. Tetany in pregnant women may or may not persist until the end of the pregnancy. The spasms which occur during lactation tend to disappear when lactation is over. Frankl-Hochwart maintains that maternal tetany has a grave prognosis.

Treatment.—As a *prophylactic* measure, it should be urged that partial resection of the thyroid glands should take the place of total excision, and that the epithelial corpuscles should specially be spared.

If the trouble is due to exposure to chill, *diaphoretic* treatment is advisable.

Treatment of the causal disease is the chief requirement. In treating gastro-intestinal disorders accompanied by tetany, the stomach-pump should be used as little as possible. Operative treatment of pyloric stenosis

[1] *N. C.*, 1903. [2] *A. f. kl. M.*, Bd. lxxvii. [3] *Jahrb. f. Kind.*, ix.
[4] *C. f. path. Anat.*, 1905. [5] *W. kl. W.*, 1906. [6] *N. C.*, 1906.

has also cured tetany in cases observed by Albu and Oppenheim, Mayo Robson, Fleiner, Dickson, and Richartz.[1] Moynihan[2] also advocated early surgical treatment. In the milder form the introduction of water by the rectum or by subcutaneous injection has a good effect. Fleiner gives the following prescriptions: rapid careful emptying and washing out of the stomach, rectal feeding (meat-broth, wine enemata, etc.), an energetic supply of fluid by means of subcutaneous injection twice a day of half a litre of sterile ½ per cent. salt solution, followed in twenty-four to forty-eight hours by two to four ounces of Vichy water every hour by the mouth, until an operation can be performed. He thinks these measures and resection or gastroenterostomy are indicated in dilatation of the stomach when, in spite of suitable diet and physical rest, the body, weight, and daily secretion of urine continue to decrease.

The intensity of the spasms may be diminished by the use of *potassium bromide*, *morphia*, and *chloral hydrate*. *Hyoscin* and *curare* have been prescribed in obstinate cases. Hoche thinks the attacks were shortened in one case by curara (0·0003 to 0·0006 g., or $\frac{1}{200}$ to $\frac{1}{100}$ grain). Kassowitz and also Hochsinger think that tetany of childhood may be cured by phosphorus! Faradic currents should never be used, but the *stabile application* of a weak *galvanic* current (to the back, over the solar plexus, the affected muscles, etc.) may be tried. *Warm baths* and *wet packs* to the twitching limbs have often a beneficial effect. Trousseau recommends the application of an icebag to the spinal column, and blood-letting.

Perspiration and salivation induced by *pilocarpin* are said to have cured one case of tetany. Voss advises diaphoresis by means of hot baths and salicylate preparations.

It must be left for the future to show how treatment of tetany after removal of the thyroid will be affected by the results of transplantation of thyroid tissue in animals whose thyroid glands have been removed, and in cases of myxœdema (*q.v.*). Since I first wrote this sentence in the first edition of this book, experiments of this kind have been made by Mikulicz-Gottstein, in my clinic (Levy-Dorn), and by Bramwell and others. We were able to report a case of recovery, which was followed by others. Románoff has also had very good results. In one of our cases the recovery has now lasted for two years. Other writers report only failures (Mannaberg). The parathyroid glands have been very much used in treatment during the last few years, and have been the means of cure in cases reported by Vassale, Marinesco,[3] Eiselsberg, and Loewenthal-Wieprecht.[4] The latter prescribed 0·02 to 0·04 g. (or ⅓ to ⅔ grain) of pure parathyroid gland per day, or 0·1 to 0·2 g. (1½ to 3 grains) of the Freud-Redlich tabloids. Experiments have been tried in transplanting normal epithelial corpuscles. Frankl-Hochwart and Pineles speak with great reserve as to the value of this organo-therapeutic measure.

Lactation should be stopped. It is very important that weak children should be well fed and strengthened by iron, quinine, or cod-liver oil.

Gregor, Finkelstein, Stoeltzner, and others have found it advisable to substitute some other food for cow's milk in children with tetany and spasmophilia.

[1] *Z. f. kl. M.*, Bd. liii.
[2] *Boston Med. and Surg. Journ.*, 1903.
[3] *Semaine méd.*, 1905.
[4] *Z. j. N.*, xxxi., and *Heilkunde*, 1907.

Chorea Minor

(St Vitus's Dance)

Bibliography in Wollenberg, Chorea, Nothnagel's "Handbuch," xii., and in the handbooks and textbooks on children's diseases.

Many different conditions go by the name of chorea. These are all characterised by marked muscular contractions and involuntary movements of a complicated nature. These conditions, however, apart from these symptoms, form a heterogenous group and should be clearly differentiated from each other.

Chorea minor,[1] also known as Sydenham's chorea, is much the most common of these affections, and is pre-eminently a disease of childhood and youth. It seldom affects infants, the great majority of cases occurring between the ages of five and fifteen. Girls are affected three times as often as boys. This disproportion is even more marked between the ages of fifteen and twenty-five, when practically only *women* are affected. After this age, chorea becomes increasingly rarer, but no age is absolutely immune. The term *chorea senilis* indicates that it may occur in old age. *Delicate, anæmic, excitable* individuals are specially predisposed to it, and the *neuropathic* constitution increases the tendency. Chorea and hysteria are therefore frequently associated, quite apart from the fact that there is a kind of hysterical twitching which is known as *hysterical chorea*. In a large proportion of cases it can be ascertained that other members of the patient's family suffer from nervous disease.

Chorea often develops without any recognisable cause. It is often thought to be due to some mental excitement, *fright* in particular, but the reason for the terror is sometimes so insignificant that its effect can only be due to the existence of marked increase of excitability. The germ of the disease has sometimes become evident before the shock which called it into full development. *Mental excitement* is a more obvious causal factor in adults than in children. A comparatively large number of those who suffer from chorea are girls between sixteen and twenty-two years of age, and in a great many of these cases I have been able to trace the onset of the disease to some mental shock.

Chorea may be produced by *imitation*. Small epidemics have been observed in boarding-schools, etc., but those affected have as a rule been hysterical individuals. The chorea produced by mental infection is undoubtedly not a true, but an *hysterical* condition.

The relation of chorea to *pregnancy* is clearly established. It appears during the first—specially the third to the fifth—months of pregnancy, in women who have been previously healthy or have had chorea in childhood. They are generally *young primiparæ*, very often unmarried. The pregnancy therefore apparently often merely increases the predisposition, the onset of the disease being due to other factors, notably mental emotion. But its relation to pregnancy is so close that it usually disappears when the pregnancy is terminated, either naturally or by premature confinement, and very often recurs in later pregnancies. It is only in very rare cases that chorea first develops in the *puerperium*.

A few observations indicate that *trauma* may be followed by chorea. I have in a few cases seen the chronic form in adults result from

[1] Chorea major or magna is not an independent disease, but an hysterical condition.

injury. A. Westphal has also observed this. *Masturbation* is a doubtful cause.

Ewald-Witte (*B. k. W.*, 1908) has seen acute chorea occur in the course of a severe gastro-intestinal disease.

The relations which exist between *chorea*, *articular rheumatism*, and *endocarditis* are of special interest. Although the views of various physicians differ widely, it is an established fact that chorea exceedingly often develops after acute articular rheumatism. In many cases an endocarditis first appears, accompanied or followed by chorea. The endocarditis may be discovered for the first time in the course of chorea, the rheumatism may occur during its course, or an old heart disease may be found in a patient under treatment for chorea. It should be noted that diseases of the heart are much more common in the *chorea of youth and pregnancy* than in that of childhood.

See also the statistics of Thayer (*Journ. Amer. Med. Assoc.*, 1906), based upon 808 cases.

Many theories have been expressed as to the nature of these relations. Some observations indicate that the *embolic* material passing from the heart to the brain and producing occlusion of the small vessels and circumscribed foci of softening may give rise to the symptoms of motor excitement. Rheumatism, however, often produces chorea without the intervention of endocarditis, and embolic processes have only been found in a small number of cases. It has also been suggested that the infective process may cause *thrombosis* of small cerebral vessels. Another theory is that the heart disease may produce the chorea in a *reflex* way, and yet another, that the chorea is due to a micro-organism or *virus*, which is capable of producing rheumatism and endocarditis as well as chorea. A number of writers (Laufenauer, Triboulet, Bechterew, Mircoli, Heubner, Wollenberg, Neumann, Frölich, Gram, Bruns, Duckworth, etc.) are inclined to think that chorea has always an *infective origin*, and that the other factors act merely as exciting causes. The evidence of micro-organisms in the brains of choreic cases, which would support this view, is scanty and uncertain (cases of Maragliani, Richter, Berkley, Dana, H. Meyer, Painese, Preobrajenski, Guizzetti, Cramer-Többen [1]). Pianese found a diplobacillus and a diplococcus, and reports that he produced experimental chorea by cultures of these. According to Maragliani's review, *staphylococci* were found in seven cases, diplococci in two, and a bacillus in two. Westphal and Wassermann,[2] in a case of severe chorea after acute articular rheumatism, found a staphylococcus in the blood and tissues, which when introduced into the blood of animals produced a condition of acute articular rheumatism. Results of this kind have also been reported by Poynton and Holmes,[3] whilst streptococci were present in the cases of Meyer,[4] Sander, and Cramer-Többen. These facts at any rate point to the infective character of chorea and to its close relationship with acute articular rheumatism. Wollenberg terms it a metarheumatic affection, and Duckworth calls it rheumatism of the brain. G. Köster established an infective origin in 71 per cent. of his cases, but recognised other additional factors.

[1] *M. f. P.*, xviii.; includes bibliography.
[2] *B. k. W.*, 1899.
[3] *Lancet*, 1906.
[4] *Jahrb. f. Kind.*, Bd. xl.

Schaps (*Jahrb. f. Kind.*, xi.) believes that the results of his examination of the blood indicate a relationship between acute rheumatism and chorea.

Chorea occasionally follows *scarlet fever*, *measles*, *influenza*, *diphtheria*, *nephritis*, *tonsillitis*, and *typhoid*.

The forms which occur in *old age* should seldom be classed with chorea minor.

If we compare the various causal factors we become convinced that chorea may be produced by *poisons of different kinds*. The age at which the motor inhibitory system is not yet fully developed and at which mental impulses may be transformed without restraint into motor actions is particularly liable to the disease. Children, young girls and women under the influence of *worry* and such mental excitement often exhibit motor restlessness which is very similar to chorea. *Individual exaggeration* of this tendency is probably a factor in the production of the choreic disposition. The condition may therefore be brought on by a severe mental shock, by pregnancy (? in a reflex manner), or finally, and most commonly, by the action of a poison produced within the organism of a person suffering from articular rheumatism or endocarditis or penetrating into it from without. (There is here a certain analogy with *epilepsy*, which, although inherited, may be brought on by mental shock, infection, and poisoning.)

Krafft-Ebing (*W. kl. W.*, 1899), Starr ("Festschrift Jacobi," New York, 1900), and others have of late carefully studied this question of the etiology, but have gained no further light upon it.

Symptomatology.—The disease usually develops from insignificant beginnings. The child becomes restless, awkward in his actions, and more apt to drop things. His teacher complains that he will not sit still, or that his writing has become untidy and slovenly. The child may at first be regarded as naughty, and his character sometimes seems to alter, but it soon becomes evident that his condition is a pathological one.

The physician is struck during the examination by his *motor restlessness*. The child neither stands nor sits still, but is constantly in motion. The arm is abducted, adducted, or rotated, the shoulder raised, the hand extended, the fingers spread out, flexed, and extended; the trunk is rotated or thrown from side to side; the forehead is wrinkled, the mouth drawn to one side, the head thrown to one side, etc. Walking becomes impossible only in severe cases in which the lower extremities are chiefly affected. The spasmodic jerkings of the legs may be so strong that the patient cannot stand. These movements succeed each other in *constant variation*, and so unceasingly that different groups of muscles and the muscles of different limbs are all in action at the same time. The characteristic feature of the condition is that the involuntary movements are not limited to one group of muscles in which they are rhythmically carried out, but that different muscle groups are all involved in an absolutely irregular manner. The various actions correspond less to the type of twitchings than of voluntary movements, but, unlike the latter, they are *purposeless* and *vary constantly in form and direction*. The combined motor acts are as a rule associated with short twitchings, *e.g.* in the facial muscles.

Foerster[1] has shown that here we have not the regulated co-operation of the antagonists with the main agonists and synergists, as in voluntary movement, but that single muscles or

[1] "Das Wesen der choreat. Bewegungsstörung," Volkmann's "Sammlung," etc., 1904.

muscle groups participate in the contraction, so that *e.g.* in extension of the finger the extensor communis digitorum acts without the interossei; in closing the fist the wrist is not extended, etc. There is therefore something unnatural and peculiar about the movements of chorea.

The *expressions of emotion*—laughing and weeping—are exaggerated and easily provoked.

As regards the *distribution* of the movements, the *upper extremities* are chiefly affected, and usually also the *muscles* of the face and trunk. The legs are less markedly involved. *The organs of articulation*, especially the *tongue*, are very often involved. The latter is twisted within the mouth or pressed between the teeth, and a more or less marked *impairment of speech* is thus produced. The words are shot out, suddenly interrupted, pronounced indistinctly, or cut up by irregular breathing movements. Smacking, clucking, gurgling noises may be caused by the action of the tongue muscles. Speech may be so gravely affected that in rare cases the patient does not utter a word for days or weeks; an actual *mutism* or aphasia is apparently present, but its choreic nature is shown by the fact that an attempt to speak brings on the characteristic choreic movements in the muscles of articulation, especially those of the tongue. The tongue is rapidly protruded and drawn back again. The involvement of the muscles of the lips and tongue and the upper muscles of deglutition in the twitchings may seriously affect the taking of food, but this does not occur in slight cases. The *respiratory muscles*, the diaphragm in particular, are usually affected; irregular spasmodic breathing is produced. Involvement of the *muscles of phonation*, causing sounds and words to be shot out, is less common. In many cases the *ocular muscles* participate in the movements; the patient cannot fix his eyes, but moves them constantly from one point to another. Transient *strabismus* may also occur.

The disturbance usually commences in *one arm* and later spreads to the other or to the leg of the same side. In many cases the chorea is entirely limited to one side of the body (*hemichorea*), or becomes general only later in its course.

The choreic contractions are as a rule increased by *voluntary movements*. The latter are only impaired if the involuntary movements accompany, combine with and modify them. The patient consequently endeavours to carry out the active movements as rapidly as possible, and to make use of the moment when the choreic contractions have subsided. His movements are therefore hurried and abrupt, and he cannot accomplish any work which requires continued co-ordinated movements or steady output of force (writing, manual work, etc.). In some cases the contractions are so slight during active movements that these are hardly affected. The numerous relations between active and choreic movements have been thoroughly discussed by Russell and Foerster.

The latter agrees with us in drawing attention to the inconstant, irregular, and uncontrolled character of choreic movements, and to the tendency to purposeless associated movements. Co-ordinated movements of the fingers, *e.g.* opposition of the thumb and index or little fingers, are usually impossible in chorea.

Emotional excitement has the greatest influence upon the muscular unrest. Conversation with the physician, or even the feeling of being observed, is sufficient to increase it greatly. In some cases the patient may succeed in controlling himself for a short time before other people,

but any emotion (such as is caused by examination, by the question whether he weeps easily, or the request to recite a poem) is sufficient to bring on the choreic movements again. It is thus evident that mental rest has a very beneficial influence. This corresponds with the fact that the contractions usually cease entirely during sleep, except in the very rare cases which I have described,[1] in which on the contrary the restlessness became most marked during sleep and entirely disappeared on waking (*chorea nocturna*).

The *intensity* of the contractions varies greatly. In some cases they are so slight as to be recognised only by an experienced observer; in others they are so severe that the whole body is involved in the wildest movements, the patient throwing himself about, flinging himself out of bed, biting his tongue, and injuring himself in many ways; he can take no food, and in his condition of *muscular insanity* (folie musculaire) he is a distressing spectacle.

Brissaud (*R. n.*, 1896) and Patry speak of a "*chorée variable ou polymorphe*," which occurs specially in degenerates. In it the contractions are very inconstant; they may be absent for days and can to a certain extent be controlled by the will, etc. They also vary greatly in intensity and character. Other observations and descriptions are given by Féré, Couvellaire-Crouzon (*R. n.*, 1899) and Moussous (abs. *R. n.*, 1902).

Involuntary movements are the most prominent and sometimes the only symptom, but in most cases the *mind* is also affected, so that irritability, depression, absentmindedness, and forgetfulness are present in addition to the motor symptoms. These mental anomalies develop—very seldom in children, but frequently in adults—into marked *psychoses*, *conditions of depression*, and in particular *hallucinatory delirium* with acute *maniacal* excitement and *confusion*, or in rare cases into a mental disturbance resembling *acute paranoia*. The *mania* usually develops at the height of the chorea, lasts for a few weeks, and then passes into depression, apathy, or a mental disorder characterised by sensory hallucinations, ideas of persecution, self-reproach, etc. The mania may, however, last for a long time (until the patient recovers or dies). The gravest types occur in the chorea of pregnancy and the infective form.

Paralysis is not a symptom of chorea; the motor power is undiminished in typical cases, although it is irregularly exerted. But there is a form in which the illness begins with a kind of *paresis* or *pseudo-paresis*, and is to some extent masked by these symptoms (Todd, West, Charcot). I have seen cases of this kind (Dissertation by Färber), and others have been described recently by Filatow, Gumpertz, Rindfleisch, etc. The relatives notice, for instance, that the child uses one arm less than the other, or ceases to use it at all. This motor disturbance may spread to both arms, the arms and leg of one side, or to the whole body, the child lying as if *paralysed* (chorea mollis, limp-chorea, paralytic chorea). He can still move his limbs to order, but only for a time and very feebly. The muscles become flaccid, the tonus decreases, and the tendon reflexes are feeble or even abolished (Gumpertz, Rindfleisch[2]). *Transient twitchings* reveal the choreic nature of the condition to a close observer. They are so slight that they have to be looked for, but they may be markedly evident in the non-paralysed muscles. In the course of the disease the twitching becomes

[1] "Dissert. Färber," Berlin, 1885, and the first edition of this text-book.
[2] *Z. f. N.*, xxiii.; with bibliography.

more marked whilst the paresis diminishes. If this choreic pseudo-paralysis affects the muscles of articulation, a kind of choreic mutism develops (Oppenheim). Charcot states that the contractions may disappear in the course of chorea, a kind of paralysis appearing in their stead.

The frequency of true pareses in the severe forms of chorea is specially emphasised by Bruns (*N. C.*, 1905). He points out that the mutism and dysphagia may persist for months and that they can often not be explained by any marked choreic unrest in the corresponding muscles and must be due to a real motor weakness. He therefore thinks one may speak of a *bulbar-paralytic* syndrome of chorea. This is in agreement with Price's observation (*N. Y. Med. Journ.*, 1907) that salivation is not uncommon in severe chorea. As regards the paresis of chorea, see also Michel (*Thèse de Paris*, 1904), and as to the speech disturbances consult Cizler (*Casop. lek.*, 1905; *N. C.*, 1905).

The muscles retain their *normal size* and electrical excitability. Muscular atrophy, which I have never observed, has been occasionally mentioned (Elloy [1]). Except in the paralytic form, the *tendon reflexes* are normal. Bonhoeffer and Oddo,[2] and also Kopzcynski,[3] state that in consequence of a hypotonia which accompanies the chorea the reflexes are diminished and sometimes even absent, but I have been unable to confirm this personally. In typical cases I have neither found any real diminution of the muscle tonus nor decrease in the tendon reflexes. Gordon has noticed that the knee-jerk has often the character of a tonic muscular contraction. Eshner [4] confirms this, and I have several times seen the same symptom, which may either be a mere coincidence or may possibly be caused by a reflex choreic contraction of the quadriceps occurring simultaneously with the reflex movement.

The *sensibility* is not affected in typical cases. Hemianæsthesia with sensory disorders is found only in hysterial chorea or in a combination of the two neuroses, but *concentric narrowing of the field of vision* apparently occurs in *simple* chorea.

Ophthalmoscopic examination does not show any change in the fundus oculi. I have never seen optic neuritis, which has been reported in a few cases. The pupils are often dilated, but they react promptly to light. Cruchet [5] has noted the frequency of hippus in chorea. The pulse is usually rapid, but seldom irregular. The sphincters act normally, except when apathy and hebetude produce incontinence of urine and fæces.

The *general condition* is only disturbed when violent muscular movements prevent nourishment being taken and thus cause exhaustion. The *temperature is increased* in severe cases, and in those ending fatally it has risen to 42·6° (109° F.).

Complications.—We have already referred to the common combination of chorea and the psychoses. It is also frequently associated with the *neuroses*. Choreiform movements may occur in exophthalmic goitre, and the two conditions may be combined (Oppenheim, Gowers, Sutherland [6]). It is specially apt to be associated with *hysteria*, and in this neurosis a true chorea may develop and run an independent course, or chorea may give rise to hysteria in predisposed individuals. In addition, there is an hysterical chorea, *i.e.* a motor neurosis arising out of the hysteria which, in spite of a great similarity to true chorea, yet differs from it in some

[1] *Thèse de Paris*, 1904; *R. n.*, 1905.
[2] *Gaz. des hôp.*, 1900.
[3] *R. n.*, 1903.
[4] *Phil. Med. Journ.*, 1901.
[5] *R. n.*, 1904.
[6] *Br.*, 1903.

essential points, viz., in its sudden onset after some mental shock, or its production by means of imitation, in the presence of stigmata and choreogenic zones, and above all in the character of the contractions, which consist chiefly in rhythmic, and sometimes violent movements of the extremities. These movements are systematic, always repeated in the same stereotyped way as if the patient were bowing, using his arms in swimming, beating a hammer on an anvil (chorea malleatoria), etc. etc. They may persist for days, weeks, months, and even longer, and then suddenly disappear after a spasm or some excitement, with the menses, etc. The movements of hysterical chorea may also have an arhythmic character. *Epilepsy* is much less often associated with chorea. Congenital mental weakness or imbecility may be accompanied by stabile chorea.

The most important complication of chorea is *endocarditis* and *heart disease*. Mitral insufficiency is the most common condition. One must, however, be very careful in chorea not to diagnose an organic disease on account of an accidental heart murmur. The patients are often anæmic, and it is therefore not unusual to hear a systolic murmur above the mitral and pulmonary valves, which is simply an *anæmic vascular murmur*. A slight extension of the cardiac dulness towards the right may also be due to anæmia and may subsequently disappear. On the other hand we must admit that the endocarditis associated with chorea is usually of a mild nature, and may disappear without leaving any trace. It seldom happens in chorea that an embolus from the diseased heart passes into the brain, and produces symptoms of paralysis, as *e.g.* in a case described by Simon-Crouzon.[1] Embolism of the central retinal artery has occasionally been observed in chorea (H. Thomson).

The articular rheumatism which develops in the course of chorea is generally of a mild character. The patient sometimes complains of pain in the limbs, for which no local cause can be discovered. Pain of this kind must, of course, not be immediately ascribed to rheumatism.

Duration, Course and Prognosis.—Chorea has an average duration of two to three months, but it often lasts longer—from six months to a year. It very seldom persists for one or more years, or assumes the character of an incurable disease. Very slight cases, on the other hand, may recover in a few weeks. Heubner succeeded by judicious treatment in lowering the average duration to six weeks.

The *prognosis* is entirely favourable, and complete recovery is the rule. There is little danger to life. So far as we can gather from the published cases, the chorea of childhood is fatal in about three, at the most five per cent. of the cases. Death occurs only in the *exceedingly severe* forms in which the muscular contractions were so excessive as to make sleep and nutrition impossible, and thus produce a condition of exhaustion which proves fatal either of itself or by means of a *fatty degeneration of the heart.* According to Richon, death is either due to a fulminating collapse without any objective cardiac symptoms, or to endocarditis, but changes in the heart are usually found even in cases of the first class. Vicq deals with this subject in his thesis (Paris, 1903). Rachmaninow[2] states that fatal chorea is usually a rapid disease which runs its course within a few weeks, and that the appearance of an exanthema resembling that of scarlet fever is an unfavourable sign. The injuries which the patient brings upon himself may prove fatal, on account of the difficulty in treating them.

[1] *Rev. mens. des mal. de l'enf.*, 1904.

[2] *A. f. Kind.*, Bd. xlv.

The age of puberty and the second decade are particularly dangerous, as cardiac defects are most common at that age. The prognosis as to life is not gravely affected by the existence of an endocarditis, as this very rarely has a fatal termination. But the results of post-mortem investigation show that death from, or in the course of chorea occurs practically only in *individuals with cardiac defects*. A fatal termination is not so uncommon in adults.

The prognosis of *chorea gravidarum* is very much graver. About 25 per cent. of the cases end fatally. Of twenty-nine persons whom French and Hicks [1] treated for this disease, three died. This is partly due to the extreme intensity which the contractions often attain, to the comparative frequency of the combination of psychoses and severe endocarditis with this chorea, and to the danger frequently entailed in this cachectic condition by abortion, spontaneous or artificial miscarriage, and even by normal delivery. If there are no complications the disease usually ends here also in recovery, which in all but a few cases coincides with the termination of the pregnancy. The mental change seldom persists after delivery. The prognosis as to life is apparently more favourable in chorea gravidarum if the patient has suffered from chorea in her youth.

Rapid emaciation, delirium, marked rise of temperature, etc., occurring in the course of chorea, are ominous signs.

We cannot definitely predict the duration of the disease, but as a rule it lasts for several months (two to three). The older the patient is the more likely is the course to be protracted. I have also noticed that chorea may linger on for a long time in weak-minded children. But even long duration does not exclude the hope of recovery. It has been known to disappear after lasting for many years. In one woman who had chorea from the age of seven to twenty-four, it disappeared with the commencement of the first pregnancy. If it develops in the latter part of life, it will probably be a permanent disease.

Chorea is a condition which tends to *recur*. It may affect the same individual several times. There are generally only one or two relapses, but cases of nine recurrences have been observed. The intervals between the attacks average about a year, but there is no regularity with regard to them. In some cases the relapses follow in such quick succession that they may be termed cases of *chronic intermittent chorea*. There is actually a *chronic perennial form*, which lasts for years or for a whole lifetime. This hardly ever appears in the chorea of childhood, which is very rarely stationary, but a "*chorea adultorum permanens*" may occur, in addition to the hereditary form which will be discussed later.

Ordinary chorea minor may appear in old age and run a typical course. Senile hemichorea ending in recovery has also been observed (Riesmann). According to a review of published cases (Bischoff [2]) it takes this course in about 20 per cent. of the cases, but as a rule *senile chorea* is a permanent condition. It is often, though not always, associated with mental disturbances.

Differential Diagnosis.—The diagnosis is in most cases very easily made. There may be some difficulty in those which commence with a kind of paralytic weakness in one arm. Spasmodic movements will,

[1] *Practitioner*, 1906. See also the Dissert. of Gettkant, Berlin, 1905, and the paper by A. Martin, *D. m. W.*, 1906.

[2] *A. f. kl. M.*, Bd. lxix.

however, be noted on careful examination, and the diagnosis is confirmed by the mental condition. It is possible to mistake chorea which has existed from early childhood for the choreic-athetotic form of infantile cerebral paralysis (*q.v.*). This may extend over the whole body, the choreic factor being so marked as to conceal the paralysis. But close examination will show the muscular rigidity, especially in the legs, the Babinski sign, the associated movements, and the athetoid character of the contractions, and confusion will thus be prevented. Moreover, the congenital or early acquired permanent form of chorea is exceedingly rare, and such cases always arouse suspicion that the motor symptoms are due to an organic disease of the brain.

In exceptional cases the chorea chiefly affects the legs and produces a disorder *of gait similar to ataxia.* In one case of this kind in a boy of three years old, I at first suspected the presence of a spinal disease until I noticed that the inco-ordinated movements were present during rest. The trouble came on after a fall, and disappeared within a few months.

Chorea is most often mistaken for *general tic.* The differential points have been discussed under that heading. There are rare cases of *partial chorea*, limited to the *muscles of speech* or those *of the eyes, lips, tongue, pharynx, and larynx.* This form gives rise to severe, though limited symptoms, and it may be of a very obstinate nature. Onodi thinks laryngeal chorea is simply a symptom of a general neurosis.

Pathological Anatomy.—As a rule only severe, complicated cases can be examined post-mortem. The cause of death is in most cases an endocarditis, a valvular defect or fatty degeneration of the cardiac muscle. The *brain* usually shows *no change*—this was the case in a recent investigation by Kopczynski—but in other cases alterations of many different kinds have been found, *e.g.* hyperæmia of the brain, hæmorrhage, foci of softening or inflammation, especially in the central ganglia, vascular disease with thrombosis, inflammatory changes in the cerebral membranes, hæmatoma of the dura mater, sinus thrombosis, etc. etc. (observations of Meynert, Dickinson, Ogle, Anton, Dana, Nauwerck, Macleod, Kroemer, Reinhold, Geddes-Clinch, Preobrajensky, Okada, Peinár, etc.). Embolism of the larger vessels with corresponding softening was seldom found; occlusion of the smaller vessels and capillary embolism of the central ganglia were rather more common. The nodules and concretions known as *chorea-corpuscles*, found in the lenticular nucleus and referred to chorea, have subsequently been found in the brains of persons who died from other diseases (Wollenberg). Hudovernig regards the formation of colloid corpuscules as characteristic. Some authors think they have found fine changes in the nerve-cells, but as these were usually Nissl changes (Daddi-Silvestrini), they may be left out of account. Abrahams states that he has seen cell infiltration of the cortex in the motor region in a case of chorea gravidarum. Reichardt describes "encephalitic" processes. Pathological conditions have occasionally been found in the spinal cord (Hutchinson, Clarke, etc.). The changes are on the one hand very inconstant and variable, and on the other they hardly ever occur except in severe and complicated cases which differ from the usual type of chorea; they are therefore not calculated to throw any light on the nature of the disease. Gowers thinks they are secondary changes. Bechterew suggests that the infectious agent does not as a rule produce gross lesions in the nervous system, although it may occasionally do so.

There is no doubt that we do not as yet know the pathological cause of chorea. It is certain that it is not as a rule caused by gross pathological changes, and that in typical cases there is either no organic disease or merely fine changes which are capable of regression. On the other hand we know that choreic contractions may occur among the other symptoms of organic brain diseases (see, for instance, post-hemiplegic chorea, p. 691). There can be no question of the fact that chorea is a brain disease, but we cannot say with certainty whether the lesions are localised in the central ganglia, the cortex, the cerebellum, or in all these. Gowers, Bonhöffer, and Foerster think the cerebellum or the tract of the superior peduncles is the starting-point of the condition.

It is desirable that we should have further investigations into the muscular changes found, specially by Rindfleisch (*Z. f. N.*, xxiii.), in paralytic chorea. A few recent observations of the pathological myo-histology with regard to the influence of prolonged forced muscular action upon the structure of the muscular tissue make this specially necessary. In a case of *senile chorea* examined post-mortem by Bischoff, the results were practically negative.

Treatment.—The extraordinary effect of mental excitement upon choreic contractions and the marked increase of excitability which characterises choreic patients is an evident indication that avoidance of all emotion and excitement is one of the most important factors in the treatment of chorea. The child should not be sent to school; the *principle of isolation* should be carried out at home, the child being kept apart from his playmates and even from his brothers and sisters, his only companions being his mother or a competent nurse. He should be kept *occupied*, but neither tired nor excited, and he should never be threatened or scolded; it is a mistake to think that chorea can be suppressed by an effort of will. If threats or punishments have the desired effect, the case is in all probability one of imitation-chorea.

The child should live in a large airy room, unless in mild cases, when he may be taken into the open air. His diet should consist of light, easily digested, but nourishing food. Coffee, tea, and alcohol must be avoided, and large quantities of milk should be given.

If the chorea is severe, the child must avoid active movement as far as possible. He should at first and for a considerable time be strictly *confined to bed*. It is particularly desirable that he should *sleep* long and soundly. If he sleeps badly, *hypnotics* should be given. If the contractions are so violent as to suggest the possibility of injury, he should be laid on a soft mattress on the floor, the walls being lined with cushions or mattresses. A *convulsion-bed*, deep and well-padded, is excellently adapted for these cases. *Confinement in an institution* is only necessitated by the presence of marked psychosis, but treatment in a hospital should always be preferred to unfavourable home conditions.

Medicinal Treatment.—*Arsenic* should be tried in every case, as this drug is of considerable value. About four drops of Fowler's solution should be prescribed for children of five to ten years old, the dose being carefully increased to eight or ten drops. Arsenious acid is more effectual and reliable, and it may be combined with iron, the dose being at first a half to one mg. ($\frac{1}{130}$ to $\frac{1}{65}$ grain), gradually increased. See p. 558 as regards the modern preparations of arsenic. Subcutaneous injections of cacodylic acid in doses of 0·02 to 0·04 g. ($\frac{1}{3}$ to $\frac{2}{3}$ grain) have been used by Lannois in severe cases with excellent results. As soon as the arsenic causes

disorder of digestion, conjunctivitis, herpes, neuritis, and other symptoms of poisoning, it must of course be stopped. The *bromides* have a soothing effect. A. Martin has recently advocated these highly in chorea gravidarum. *Chloral hydrate* may be beneficial in bad cases. The production of " prolonged sleep " by its means has even been advised. *Chloroform* should only be used in the severest cases, but it should be remembered that the heart is apt to be weak in such cases, and its continued use may therefore be dangerous. *Subcutaneous injections of chloroform* have proved of service in a few particularly severe adult cases under my care. *Antipyrin*, given carefully in several doses of 0·3 to 0·5 g. (5 to 8 grains), has sometimes a good effect. Comby has given it in larger doses without any unpleasant consequences. Carrière and Leclerq give it in daily doses of 6 to 9 g. (90 to 140 grains) (!) and more. That this should lead to a cure within ten to fourteen days seems to me doubtful. Comby, moreover, regards arsenic as a more powerful remedy. *Physostigmin* has been recommended. Zinc (valerianate, oxyd), conium, cannabis indica, etc., have proved to be of no use. Exalgin, preparations of salicylate (salol, salophen, etc.) and ol. Gaulther. procumb., etc., have all been recommended of late years. Bourneville-Noir and Bossard have found monobromated camphor good, in small but increasing doses. Moncorvo reports success from the use of analgen (2 to 6·0 g. (30 to 90 grains) per day). Randlcy found hyoscin. hydrobrom. useful in obstinate cases. Adams had been successful with small doses of sulphonal and trional, and Ziehen with chloralamid (in chorea gravidarum).

Mild hydrotherapy, in the form of simple washing or partial rubbing with cold or warm water, should be tried in every case. Warm half-baths lasting for one to two hours have sometimes been beneficial. Heubner combined diaphoresis with arsenic and rest in bed. Electrical treatment is of doubtful benefit, but some physicians have found galvanism to the back, head, and to possible pressure points, and electric baths of service. The " inhibition-exercises " have been helpful in many of my cases. Breitung has induced recovery by the removal of a foreign body from the ear, and R. Müller by opening the mastoid in ear disease, but such cases can hardly have been ordinary chorea minor. Hypnosis has been said to be successful in rare cases. Bier's attempt to cure the condition by congestive hyperæmia produced by ligature, and Jemma's [1] advice to employ lumbar puncture, should merely be mentioned on account of their novelty.

The same principles should be followed in the treatment of adult chorea. In the chronic form of the disease, treatment will naturally have little effect. In adults even more than in children, the mental condition should be borne in mind, and isolation should be tried in most cases.

Huygh's method (*R. n.*, 1903) of immobilising the limbs under chloroform is certainly not suited for true chorea, and should only be tried with great care and discrimination in the hysterical form.

Chorea gravidarum makes special demands upon the physician. The fœtus must always be considered in prescribing remedies (hydrotherapy, arsenic, etc., must therefore be very cautiously used). The condition is often greatly aggravated by comparatively slight excitement. Artificial termination of the pregnancy may become *necessary to save the patient's*

[1] *Gaz. degli Osped.*, 1901.

life. There can be no hesitation about this course if the child is viable, but on the other hand it should only be resorted to if life is endangered by the intensity of the contractions, a condition of exhaustion or of heart disease, nephritis, psychoses, etc. The subject has been recently discussed by A. Martin (*loc. cit.*) and Binswanger.[1]

In convalescence, *gymnastic* exercises are often of great value. Roth,[2] Bruel, and others agree with me on this point. A stay in the country, at the sea-side or in the hills, may help to establish the cure.

Other Forms of Chorea

Hereditary Chorea (Chorea Chronica Progressiva, Huntington's Disease, Dementia Choreica)

This must be regarded as a disease *sui generis*, and differentiated absolutely from chorea minor.

It is, on the whole, a rare disease, which is transmitted from one generation to another. One generation may be passed over, or may suffer from epilepsy and hysteria in place of chorea. The published cases seem to indicate that if one member of the family is spared, his children may be free from the disease.

Men and women are equally often affected. The disease usually occurs between the ages of thirty and forty, but it may develop much earlier or later. It rarely commences in youth or old age. When it descends through several generations it shows a tendency to develop later and later (Heilbrönner[3]). It may occur without any exciting cause, or may develop after some mental excitement, the puerperium, etc.

The cardinal symptom of this condition is motor restlessness. This is at first of slight intensity and limited to certain areas, *e.g.* the face, upper extremities, etc., but in the course of years it increases in severity and extent, and finally involves all the muscles under voluntary control. In most cases the ocular muscles are spared. The condition greatly resembles chorea minor. There is a constant varying play of involuntary, inco-ordinated, purposeless movements in the various muscle groups. They give rise to an almost uninterrupted series of *grimaces* and *gesticulations*, and affect the speech, which is interfered with by clicking of the tongue, smacking movements of the lips, or by spasmodic inspiration. These contractions usually, although not invariably, cease during sleep, and are greatly intensified by emotion. On the other hand almost every observer notices that the patient can for a certain time suppress these choreic movements by an *effort of will* or by the execution of *voluntary movements*. He is therefore able to grasp an object, to thread a needle, to write, and to eat. These actions do not intensify, but *inhibit* the involuntary movements. This voluntary control of the restless limbs is often gained only at the expense of increased activity in the other muscles, and the voluntary movements are carried out, not steadily, but only at certain times, the chorea re-asserting itself in the intervals.

The patient is able to walk until the late stages of his illness, but his gait is altered in a peculiar way. As the choreic contractions affect in turn the muscles of the foot, the pelvis, trunk or arm, they may produce a rocking movement of the trunk, a clown-like advancing of the upper

[1] *D. m. W.*, 1907. [2] *Z. f. diät. Therap.*, viii. [3] *A. f. P.*, xxxvi.

part of the body, flinging movements of the arms, or skipping movements of the legs, or the patient may suddenly come to a standstill after taking a few steps. All these movements are constantly and rapidly performed.

The motor power remains practically normal during the whole course of the illness, although in a few cases paralysis of the hemiplegic type may develop in its later stages (Liebers [1]). The sensibility and the sensory functions are unimpaired. The tendon reflexes are usually slightly exaggerated. There are no changes in the internal organs, including the heart.

Mental changes, in particular *mental weakness* which progresses slowly and may develop into idiocy, are almost constant symptoms. Kattwinkel [2] thinks that the condition is rather one of absent-mindedness or inability to concentrate the attention than of real dementia. This factor certainly plays a part, as in almost every other motor neurosis, but there can be no doubt that grave mental weakness often develops in the further course of the disease. The patient is often depressed, and so tired of life that he may attempt to commit suicide. Increased irritability is sometimes present and may develop into intense excitement, but in the later stages it usually gives place to *apathy*. The mental weakness generally comes on some years after the motor symptoms, but it may precede them (A. Westphal [3]).

The *prognosis* is very grave, as the disease is incurable. The patient finally becomes bedridden and succumbs to some intercurrent disease, to cachexia resulting from the want of nourishment, or in a comatose condition. The condition lasts for ten to thirty years.

Evidence of *direct heredity* is of special importance as regards the diagnosis from other forms of chorea. This is extremely rare in simple chorea, and when it occurs is only found in one parent, not in whole generations. If the hereditary nature of the condition cannot be ascertained—and this may undoubtedly occur—the symptoms are not always sufficient in themselves to distinguish the disease from simple chorea. The later course, however, soon reveals the chronic, progressive form. This, of course, is also found in most cases of senile chorea. There are, no doubt, rare cases of chronic progressive chorea in which the intelligence is not notably impaired.

Böttiger, F. Schultze, and others would regard Unverricht's myoclonia as similar to hereditary chorea, but this is quite unjustifiable.

Choreiform symptoms sometimes occur in paralytic dementia, but the special symptoms then establish the diagnosis (Dräseke, *D. m. W.*, 1905).

Pathological Anatomy.—Changes in the meninges, viz., cloudiness, external hydrocephalus, and hæmorrhagic pachymeningitis, are found in many cases. These are practically always accidental and are not the cause of the disease. In two cases which I was able to investigate carefully (along with Hoppe [4]) I found *disseminated, miliary, encephalitic foci*, especially in the cortex and the motor region. Similar changes had been described previously by Greppin, and have been since found by Kalischer and Kronthal, Facklam,[5] Kéraval-Raviot,[6] and others, but in the cases examined by the latter writers there was chronic diffuse meningoencephalitis with secondary atrophy of the cerebral cortex. Binswanger

[1] *C. f. N.*, 1905. [2] *A. f. kl. M.*, Bd. lxvii. [3] *C. f. N.*, 1905.
[4] *A. f. P.*, xxv. [5] *A. f. P.*, xxx. [6] *Arch. de Neurol.*, 1900.

mentions that the changes in the cases he examined did not differ essentially from those in paralytic dementia. Lannois and Paviot[1] think that their researches show that proliferation of the neuroglia and infiltration of the cortex with glial cells are the chief changes (" malformation tératologique de la névroglie "), and they attribute to them a very important part in the origin of hereditary familial nervous diseases. Kattwinkel describes atrophy of the supra-tangential fibrous layer and the radiating fibres in the central convolutions, and accumulation of round cells round the pyramidal cells of the cortex. This cell infiltration has also been found in a recent case by Lannois-Paviot-Mouisset, who regard it as the essential change. See also Rossi,[2] Buck,[3] Spiller,[4] and Besta.[5] The latter attaches most importance to the vascular changes. It is still an open question whether these changes represent the anatomical cause of hereditary chorea.

Stier (*D. m. W.*, 1902) and Müller (*Z. f. N.*, xxiii.) think the disease is due to *congenital malformations* of the motor cortex. On this basis the subsequent changes develop, viz., the diffuse or disseminated glial proliferation, especially in the layer of the small and medium pyramids, the secondary vascular process, the atrophy of the tangential fibres, etc.

The *treatment* is symptomatic. Arsenic has no effect in the majority of cases, but in rare instances improvement has been ascribed to it.

A very peculiar form of familial chorea has been observed by Remak and myself. Two boys, sons of a woman suffering from chronic hemichorea, became affected at the age of eight with chronic progressive chorea, which commenced in the lower extremities, in one in a form resembling athetosis of the leg. This gradually spread to other muscles, and in a few years the boys showed the typical condition of general chorea, the legs being most severely affected. In one case walking was almost impossible, and in the other greatly impaired, the patient being able to walk backwards much better than forwards.

Electric Chorea

Various conditions, some of them very obscure, have been described under this name.

First of all there is a disease observed in northern Italy and described by Dubini. It may appear at any age. For a short period there is pain in the head, neck, or back. This is followed by short, rapid, muscular contractions, like those elicited by electric stimulation. They involve first one arm and one side of the face, pass subsequently to the leg of the same side, and finally to the other side. There are also epileptiform attacks, which may be limited to one side of the body. In the further course of the illness, paralysis develops, commencing in the extremities first affected by the contractures. The paralysis becomes general and is associated with atrophy and disturbances of the electrical excitability. The skin is hypersensitive, and any touch may produce violent contractions. The disease is a painful one. The temperature may be markedly raised. The mind is not affected. After some days, weeks, or months, death occurs in coma or from paralysis of the heart. Only a few cases recover. Nothing is known as to the nature of the condition. It is assumed that it is due to some infective material, and this is in accordance with the clinical observation and the post-mortem appearances in a case described by Bonardi.

Under the name of electric chorea, Bergeron describes an affection which he has observed in children of seven to fourteen years of age, those particularly of anæmic and irritable constitution. The chief, indeed the only symptom, is violent twitching, coming on in spasms and caused by forcible contractions in certain groups of muscles. Thus the head is thrown from side to side, the shoulders shrugged, the upper arm abducted, the forearm strongly flexed, etc. The movements seem as if they were produced by a rhythmical electrical stimulation. They are limited to one extremity, to single muscles, or involve many muscles of the body. They disappear during sleep. They cannot be controlled by the will; on the contrary they are increased

[1] *R. n.*, 1901. [2] *Riv. speriment.*, 1904. [3] *Journ. de Neurol.*, 1904.
[4] *Journ. Amer. Med. Assoc.*, 1905. [5] *Riv. speriment. di Freniat.*, xxxi.

by any attempt to repress them. All the other functions are normal. The prognosis is excellent, as recovery always takes place within a few days or weeks from the use of arsenic, cold douches, or an emetic (tartrate of antimony). The disease has been thought to be caused by some gastric irritation. It may be difficult to differentiate this motor neurosis from hysterical chorea.

Henoch, under the name of electric chorea, has described a form of the chorea of childhood which differs from the ordinary form in the lightning-like character of the contractions. These specially affect the muscles of the neck and shoulders, and occur at intervals of about three to five minutes. So many conditions of different kinds bear the name of electric chorea that, until further knowledge is gained, it ought to be abandoned. It has been assumed, and has lately been emphasised by Cade and Fischer, that the disease described by Henoch is identical with myoclonia. Bruns (*B. k. W.*, 1902) thinks the forms of Bergeron and Henoch are the same. He would differentiate between a hysterical, an epileptic, and a true form of electric chorea, the latter being allied to general tic. See also Fischer, *Gaz. des hôp.*, 1903.

Paralysis Agitans

Shaking Palsy (Parkinson's Disease)

Bibliography: Parkinson, "An Essay on the Shaking Palsy," London, 1817; Charcot-Vulpian, *Gaz. hebd.*, 1861; Ordenstein, "Sur la Paralysie agitante," etc., Paris, 1868; Joffroy, *Gaz. des hôp.*, 1871; Charcot, *Gaz. des hôp.*, 1878; Westphal, *Charité-Annalen*, iii. and iv.; Charcot, "Klin. Vortrag.," German Ed., 1874, and "Polikl. Vortr.," Bd. i.; Boucher, *Thèse de Paris*, 1877; Magnan, *Gaz. méd. de Paris*, 1880; Buzzard, *Br.*, 1882; L'Hirondel, *Thèse de Paris*, 1883; Berger, "Eulenb. Realenzykl.," first edition; Eulenburg, Ziemssen's "Handbuch," xii.; Heimann (under Oppenheim), "Inaug.-Dissert.," Berlin, 1888; Siotis, *Thèse de Paris*, 1886; Lacoste, *Thèse de Paris*, 1887; Blocq, *Thèse de Paris*, 1888; Vincent, *Thèse de Lyon*, 1888; Müller, *Charité-Annalen*, xii.; Schultze, *V. A.*, Bd. lxviii.; Fürstner, *A. f. P.*, xxix. and xxx.; Berbez, *Gaz. hebd.*, 1889; Moncorgé, *Lyon méd.*, 1891; Bidon, *Rev. de Méd.*, 1891; Gowers, "Handbuch d. Nerv."; Dana, *N. Y. Med. Journ.*, 1893; Redlich, *Obersteiner*, 1894; Grawitz, *D. m. W.*, 1894; Fuchs, *Z. f. kl. M.*, xxv.; Schiefferdecker-Schultze, *Z. f. N.*, xxv.; Brissaud, "Leçons sur les malad. nerv.," 1895; H. Oppenheim, *Journ. f. P.*, i.; Krafft-Ebing, *W. kl. W.*, 1899; Frank, *M. f. P.*, 1900; Gilli, *Thèse de Paris*, 1900; Erb, "Die deutsche Klinik," etc., 1901; Compin, "Étude clinique des Formes anormales de la maladie de Parkinson," *Thèse de Lyon*, 1902; Clément, *Lyon méd.*, 1902; Collet, *Lyon méd.*, 1903; Huet-Alquier, *R. n.*, 1903; Alquier, *Thèse de Paris*, 1903; Hart, *Journ. Nerv. and Ment. Dis.*, 1904; H. Oppenheim, *D. m. W.*, 1905; L. Bruns, *N. C.*, 1905; Lamy, *Traité de Méd.*, 1905; Souques, *R. n.*, 1905; Catola, *Riv. di Pat. nerv.*, 1906; Camp, *Journ. Amer. Med. Assoc.*, 1907; Klieneberger, *M. f. P.*, xxiii.; Wollenberg, Nothnagel's "Handbuch," xii.

Paralysis agitans is a disease of *old age*. It usually begins during the sixth decade, but often also in the fifth. It rarely occurs before the age of forty, although I have seen it in a man of thirty-two years of age, and it has occasionally been observed in youth, by Duchenne once at the age of sixteen, by Ballet-Rose [1] at fifteen, and by Lannois [2] in a child of twelve.

Its onset in youth is an extremely rare occurrence, and such cases ought to be diagnosed with great reserve. They possibly represent a special type. A case in point is that described by Weil and Rouvillois (*Rev. des mal. de l'enf.*, 1899), with an onset at the age of ten, and which seems to me exceedingly doubtful.

In the majority of cases there is no ascertainable *cause*. The influence of *mental emotion* and *injuries* is most definitely established. Persistent worry, and more often a violent fright, are regarded as causes. Injuries to the head, trunk, or limbs, especially bruising and laceration of the nerves, may give rise to the disease, which then usually commences in the injured part of the body. In Krafft-Ebing's statistics trauma was the cause only

[1] *R. n.*, 1904. [2] *Lyon Méd.*, 1894.

in seven out of a hundred cases, but this writer attributes great importance to *physical over-strain*, and emphasises the tendency of the disease to commence in over-fatigued muscles.

See also Ruhemann (*B. k. W.*, 1904) as regards the traumatic etiology.

Heredity is not marked, although it is a predisposing cause in a good many cases.

I know one family in which two sisters developed paralysis agitans at a comparatively early age, a third becoming affected in advanced life by senile dementia with peculiar choreiform contractions of the tongue. In another of my cases the brother of the patient also suffered from paralysis agitans, and a sister apparently from bulbar paralysis. I have since known another family in which three sisters were affected.

Clerici-Medea (abs. *N. C.*, 1899) report having seen two sisters of one family develop paralysis agitans at the ages of twelve and twenty-eight, two other members of the family being probably afflicted with the same disease. Bury also speaks of a tendency to affect certain families.

Some of my cases would seem to indicate that it has a special tendency to appear in long-lived families.

It is doubtful whether paralysis agitans is related to *acute* infective diseases. In seven of my cases there had been *syphilitic* infection, and in three of these the disease appeared at a remarkably early age. Anti-syphilitic treatment had, however, no effect.

Symptomatology.—The disease is characterised by the following striking symptoms: 1. *tremor*; 2. *constant muscular rigidity*, which causes a *peculiar position of the head, trunk, and extremities*; 3. *impairment and retardation of the active movements*; and 4. a *peculiar modification of the gait.*

The tremor chiefly affects the extremities, especially the upper limbs, and in these mainly the peripheral parts—the hand and fingers. It consists of *rhythmic* oscillations, which follow each other so slowly that only about four to five (seldom more) occur in the second; these take the form of flexion and extension, abduction and adduction of the fingers; flexion and extension, pronation, and supination of the hands. The excursion of the movements is usually within narrow limits; thus the thumb and index-finger are rubbed together as in crumbling bread, rolling pills, etc. The tremor movement of the hands is more ample in comparison, and may sometimes amount to true shaking.

The outstanding characteristic of this tremor is that it *continues during rest.* The tremor is always present, whether the patient is lying down or standing, whether his hands are supported or hang by his side. *Spontaneous remissions* may, indeed, occur; the tremor ceases for some moments or even longer, or passes to other muscles, but it is never brought on by active movements. I shall refer later to the rare exceptions to this rule.

There is a remarkable *symmetry* in the movements. The same movements, *e.g.* flexion and extension of the fingers, take place in the same regular time and with the same amplitude of oscillation. This gives rise to the stereotyped character of the tremor. It may from time to time increase or diminish in intensity, but for the space of one or more seconds the oscillations remain practically the same.

Active movement stops the tremor for a time. It ceases momentarily if the patient extends his shaking hand, or alternately opens and closes it.

In forced active movements and in such continuous movements as writing, it persists or becomes increased, the writing showing the effect of the tremor (Fig. 419). The writing is also characterised by its smallness. The tremor may be increased by active movements which are associated with excitement, *e.g.* in a clinical demonstration. The patient himself tries to procure a momentary cessation of the tremor by means of the inhibiting effect of active movements ; he grasps some object, or changes the position of his limbs. He is not unusually much more comfortable when walking than when sitting or lying down, and may be able to walk long distances. In one advanced case the patient could play billiards by the hour, without being annoyed by the tremor which was marked during rest.

Passive movements may stop the tremor for a time or even altogether (Oppenheim), but it is sometimes increased in the extremity which is not being manipulated, and is therefore to some extent transferred to other muscles. I have even observed the mere attempt to touch the shaking limb, or a sudden approach to it, momentarily inhibit the tremor. This result can sometimes also be attained by concentrating the patient's attention, *e.g.* making him gaze at a fixed point. This effect is temporary and not of practical value. The tremor is always aggravated by *mental excitement*. For this reason it is always more marked when the patient

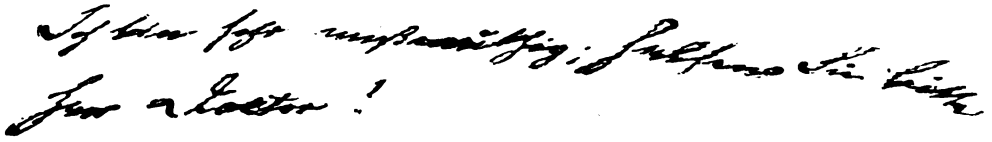

FIG. 419.—Writing of a patient with paralysis agitans.

is in company than when he is alone. Conversation, some sad recollection, etc., has the same effect.

The tremor may be limited to one arm or hand, or affect both upper extremities, an arm or leg of the same side, or all four extremities. In the hemiplegic form the rigidity may extend to the facial muscle of the same side, causing marked asymmetry of the face. When the lower limbs are affected, there is usually extension and flexion, adduction and abduction of the foot, but the muscles of the thigh are often also involved. On the whole marked tremor is less common in the legs than in the arms. The *head* is not always spared ; I have seen nodding or rotation movements in a good many cases. Tremor of the *lower jaw*, the *muscles of the chin*, *lips*, and *tongue*, is not uncommon.

Brissaud-Meige (*R. n.*, 1905) and Carrayrou (*Thèse de Paris*, 1903) mention tremor of the eyelids. In one of my cases this was so intense as to prevent ophthalmoscopic examination being made.

The *muscles of the vocal cords*, the respiratory and abdominal muscles, are rarely affected. The tremor usually ceases during *sleep*, but this is not an invariable rule. It often becomes more violent at bedtime and makes it very difficult for the patient to rest.

Chronic muscular rigidity is a valuable diagnostic symptom, especially as tremor may be absent, and it may be the only symptom. The tension chiefly affects the muscles of the *throat*, *neck*, and *spinal column*, but may involve those of the extremities and face, in fact, more

or less all the muscles of the body. It affects the active movements, but more especially it causes certain *alterations of posture*. These may be the first sign of the disease, and are often markedly evident at a stage when the passive movements are not notably impaired. The head is usually inclined forwards, less often to one side, and very rarely towards the back (Westphal, Bidon). The trunk is practically always bent forwards, the great majority of patients holding themselves in a stooping position (Fig. 420). The arms are slightly abducted, and the elbows flexed at an obtuse angle (rarely at a right or acute angle); the wrists are generally over-extended and the fingers slightly flexed, either in all the joints or in the metacarpophalangeal joints, whilst the middle and terminal phalanges are extended (*interosseous* position, *claw hand*, writer's position, etc.). The legs are either held in the ordinary position or slightly flexed at the hip- and knee-joints, the thigh being to some extent adducted.

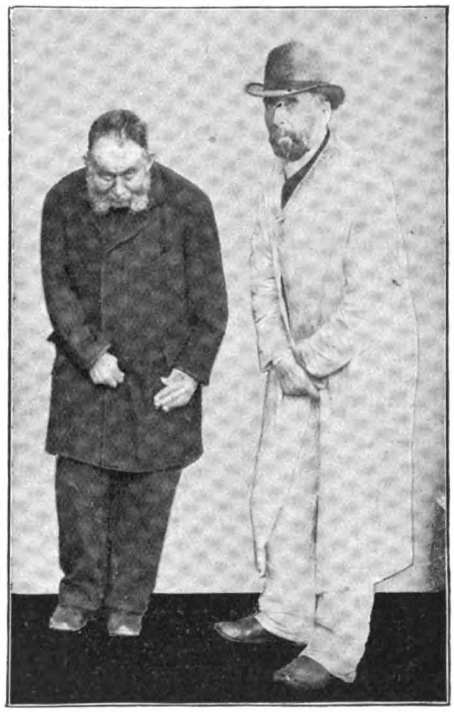

FIG. 420.—Attitude of the body in paralysis agitans. (Oppenheim.)

The muscular rigidity also gives rise to a peculiar *statuesque rigidity of the face* and the *whole body*. This is particularly noticeable when the tremor is absent, and one is reminded of a wax figure. The face is motionless; the free play of movement is absent; the facial expression, attitude of the body, position and attitude of the limbs are all changed. We see at a glance, however, that the muscles are not paralysed. The mouth is not open, nor drooping at the corners. The face has a stone-like, rather than a flaccid look, but the eyes are alive and reveal the activity of the mind.

Resistance to passive movements is always present at a late stage, and sometimes even quite early. It differs from spastic rigidity in that it is not increased or produced by passive movements, but is constant and regular, and that slow passive movements encounter the same resistance as quick movements. The head cannot be fully rotated, flexed, or extended. In some cases it seems to be absolutely fixed, and can be very slightly moved. In attempting to adduct the arm, the pectoralis major and the latissimus dorsi contract. The flexed fingers can be extended until the later stages, but they quickly resume the flexed position, etc.

Impairment and retardation of the active movements is at first a result of the muscular contraction, but as it may occur at a stage when the latter is absent, it must be considered to some extent as an independent symptom. The active movements (especially in the fingers and toes) are

retarded and less agile than before. The muscles do not immediately respond to the will. This change is apparent even in simple movements. If we ask the patient to run his fingers as he would over a piano, the movements are sluggish, especially in the hand most affected by the tremor. I have found this symptom in almost every case, and think it of value in diagnosis on account of its early development. But the impairment and retardation is naturally most noticeable in *combined motor actions*, *e.g.* dressing and undressing, rising from a seat, changing the position, etc. The patient cannot turn himself all of a piece, but does so slowly and bit by bit.

The *gait* undergoes a peculiar modification. The patient begins to walk slowly, then as a rule moves rapidly forward, as if he were about to stumble at every step (*propulsion*). There is sometimes a tendency to run backwards (*retropulsion*). This retropulsion takes place if the patient bends too far back, as in trying to reach some object above him (off a shelf, etc.); he runs backwards until he is brought up by striking some immovable object, such as the wall or a table. This can be easily brought on by pulling the patient backwards by his coat. Lateropulsion is rare.

All these disturbances, it seems to me, may be due to the fact that such a patient has special difficulty in *bringing the groups of muscles which have been in a condition of rest or tonic contraction rapidly into one of contraction or relaxation.* He cannot arrest the motion, and is compelled to continue the movement in the direction once taken. If he could quickly bring the trunk forwards, he would not be compelled to run backwards in this way. Other explanations have been attempted (Pitres, etc.).

I have seen a few cases in which the disturbance of gait, combined with a kind of basophobia, was the first, and for a long time the only symptom. See my papers in the *D. m. W.*, 1905.

Paralysis occurs rarely, and only in the last stages. I have found it present in some advanced cases, such as we see in a hospital for incurables, but it is never absolute, as a certain amount of motility is always retained.

The data as to localised paralysis, with or without atrophy (Berbez, Lacoste, Moncorgé, etc.), are too scanty to permit of their being discussed.

The *tendon reflexes* are as a rule normal; they are never absent, but are often exaggerated, though not to the extent of clonus. Babinski's sign is also absent. There may be occasionally a *false foot-clonus* (see p. 9). If, for instance, the ankle is put into the position of dorsi-flexion, tremors appear after a time in the extensors of the foot and toes (slightly, if ever, in the calf muscles). This is the tremor of paralysis agitans itself, produced in this way. The *paradoxical contraction* (see p. 62) may often be elicited, especially in the extensors of the foot, but also in other muscles, if their points of insertion are approached to each other. I should mention, however, that I have succeeded in eliciting true foot clonus in some exceptional cases. If the Babinski or Oppenheim signs are present, we must be dealing with a symptomatic form of the disease or with a complication.[1] Huet-Alquier and others regard exaggeration of the deep reflexes as the usual condition. I have several times found *abnormal associated movements* present, *e.g.* if the patient tried to move the toes of the affected limb, associated movements appeared in the toes of the unaffected foot (this was slight or absent if the sound toes were moved).

[1] One can imagine that the dorsal reflex may be produced by individual preponderance of the muscular rigidity in the flexors of the toes. Cestan-Lesours and Huet-Alquier also think that the toe reflexes are normal as a rule.

Frank has confirmed these observations from my cases. The muscles remain normal in size and electrical excitability.

Negro and Treves (*Arch. ital. de Biol.*, xxxvi.) found changes in the curve of the muscular contraction in the form of diminution of the frequency of the oscillations, and they observed peculiar muscle waves, *e.g.* in the triceps, in active movements of the muscle.

The other symptoms are less important, but some of them should be noted. *Speech* is often affected. The articulation is not impaired, nor is there any scanning, but the voice is very monotonous, not properly modulated, weak, and sometimes whining. The patient has to prepare for speaking ; it is often some time before he produces the first sound, but the words then follow each other rapidly, and may even be explosively shot out. But this speech disturbance is not a constant symptom.

For the laryngeal symptoms the reader should refer to F. Müller, *Charité-Annalen*, xii ; Rosenberg, *B. k. W.*, 1892 ; Felix, *Sem. méd.*, 1900 ; Cizler, *Casop. lek.*, 1903, and *Arch. bohém.*, 1905 ; also Mosse, *B. k. W.*, 1906.

Bulbar symptoms, in particular dysarthria and dysphagia, have occurred in rare cases (Bruns, Oppenheim, Compin, Mackintosh, Souques). Bernhardt [1] once noted forced laughter. There is never aphasia, syllable stumbling, stuttering, etc.

Hyperidrosis is not uncommon, and I have found that *salivation* may be a troublesome and early symptom. In one case this was associated with excessive mucous discharge from the nose.

For the explanation of this symptom I would refer to my paper and to that of Catola, with whom I cannot agree.

Frenkel [2] thinks he has found thickening of the skin, which I have very rarely observed. " Main succulente," glossy skin, erythema, œdema, etc., are occasionally mentioned (Vincent, etc.).

The joints are not usually affected, although French writers (Gilli) have suggested that there may be true " arthropathies parkinsoniennes." Hecker (*N. C.*, 1906) seems to have observed a similar condition, but it may have been a combination with arthritis deformans.

The functions of the bladder are not generally affected, but many conditions which impair these functions (hypertrophy of the prostate, etc.) may occur in old age. I have seen a few typical cases in which strangury and even incontinence seemed to be symptoms, and in one there was occasional incontinence of fæces, a symptom also reported by Carrayrou. Phosphaturia seems to be very common. The optic nerves are never affected—König's case must have been due to a complication—and there is never true paralysis of the ocular muscles nor nystagmus. I have only once seen *paralysis of convergence*, which was possibly simulated by tonic contraction of both abducens muscles. Saint-Léger [3] reports oculomotor paralysis. Slowing of the eye movements, difficulty in moving the eyeballs, and " lateropulsion oculaire " have also been described (Debove,[4] Neumann [5]). In two of my patients who had hemilateral paralysis agitans, oculo-pupillary symptoms developed on the affected side. I have once seen Graefe's symptom on one side in unilateral paralysis agitans. König [6] describes a spasm of accommodation. In opening the eyes the

[1] *A. f. P.*, Bd. xxxviii. [2] *Z. f. kl. M.*, 1899. [3] *Thèse de Paris*, 1879.
[4] *Thèse de Paris*, 1877. [5] *Progrès méd.*, 1878. [6] *Bull méd.*, 1893, and *N. C.*, 1894.

frontal muscles sometimes remain in a condition of tonic contraction, so that the folds on the forehead only gradually disappear (Moczutkowsky [1]). I have not personally found this.

The *sensibility* is unimpaired. Some sensory disturbance has been found in a few cases (Ordenstein, Berger, Heimann, Holms), but it is very doubtful whether this was a symptom of paralysis agitans, a complication or a symptomatic form of the disease. Karplus [2] alone found sensory disturbances (hypæsthesia and hypalgesia of the extremities). Naumann [3] found diminution of the pressure sense. I have known some rare cases which showed slight disturbances of sensibility, but am convinced from personal experience that anæsthesia is not a symptom of the typical disease.

The intelligence is, as a rule, unimpaired. The mental disorder which has developed in a few cases (Ball, Wille, etc.) should be regarded as a complication. The distressing symptoms of the disease, especially towards the end, naturally make the patient very depressed, fretful, inclined to weep, etc. Conditions of anxiety and great restlessness are specially apt to develop. When this is not so, a certain *bonhomie* may be present, which contrasts strangely with the marked physical troubles.

Grawitz has observed gastric disorders in the prodromal and early stages, and I have also noted these. One of my patients suffered for a year from severe attacks of præcordial pain with constipation. These were followed by the first symptoms of paralysis agitans. In a case of Raymond's (*R. n.*, 1905) jaundice was present at the onset of the disease.

The *subjective symptoms* are chiefly due to the muscular contraction and the limitation of the active movements. These trouble the patient most during night; his inability to change his position at will annoys him and makes him dependent upon others. *Pain* is not always present, and is not usually acute. It is generally described as rheumatoid, and may be an early symptom. It is seldom a marked symptom throughout the course of the disease ("forme douloureuse" of L'Hirondel, etc.). Vertigo occurs only in rare cases, such as those of Charcot, Vulpian, and Bruns, but the patient often complains of a distressing *sensation of heat*; he feels as if hot water were being poured over his whole body. This feeling is very troublesome during night, more especially if accompanied by excessive perspiration. A feeling of cold is less common. An actual rise of temperature may possibly occur (Fuchs).

Development and Course.—The development is usually *slow*, an acute onset being quite exceptional. In such cases we have the history of a sudden jerk passing through the arm or the arm and leg of one side, which seems to paralyse the limb. The impairment of movement and the tremor date from that time. On the other hand, paralysis agitans may follow true hemiplegia, and be confined to the limbs first paralysed, but this rare form should be distinguished from the ordinary type. This symptomatic tremor, resembling that of paralysis agitans, has several times been noticed in tumours of the cerebral peduncles (Charcot, Benedikt, Blocq-Marinesco; see pp. 695, 697). I have seen it in encephalitis of this region. This post-hemiplegic form of paralysis agitans appeared a few hours after the attack in a case described by Lamy.

The disease usually commences in one *arm*. The tremor is the first symptom noticed. It is at first slight and remittent, then gradually

[1] *N. C.*, 1897. [2] *Jahrb. f. P.*, xix. [3] *N. C.*, 1903.

increases in intensity, becomes more constant, and extends to other muscles. Slowing of movements then becomes evident. The arm assumes a forced position, and the muscular rigidity appears in the parts exempt from the tremor. A year and more may pass before the tremor extends to the other limbs, first to the other arm or the leg of the same side, finally involving more or less all the muscles of the body. In some cases a leg is first affected, then the other leg or the arm of the same side.

Tremor is not always the first symptom; slowing of movement and muscular rigidity may first attract notice. The diagnosis may then be a matter of great difficulty, so long as the symptoms are limited to one arm or the arm and leg of one side, and simulate a slowly developing hemiplegia. The disease may continue to develop without showing any tremor; the attitude of the body and the position of the extremities are all as described above, but the tremor is absent. There is paralysis agitans sine agitatione. These are the cases in which there is sometimes an *exception* to the rule with regard to the tremor; it is absent during rest, but appears, though often but slightly, in movement, *e.g.* in extension of the hand or spreading the fingers or toes. It is then generally of another character.

The course is always *very chronic*. Fifteen to twenty years and more may pass before the patient becomes quite bent and confined to bed. A patient of this kind, whom Westphal described some thirty years ago, is still alive and able to move about, but the case is of course atypical. The tremor may for many years be confined to one limb. In a case observed by Thomayer,[1] slight tremor of the foot was the only symptom for five years. Marked remissions are rare. I have once seen the symptoms greatly improve in the course of an attack of jaundice. In a number of cases which I have seen during recent years, marked and prolonged improvement was brought about by treatment. The trouble seldom progresses rapidly. If the contracture has reached its maximum, the tremor may become more and more limited. The patient is in the end entirely dependent upon his nurses, and it is difficult to keep him clean. In one neglected case under my observation the fingers were so firmly closed that the nails had grown fan-like continuations as long as the fingers. Dejerine mentions that the fingers or nails may grow into the palm of the hand (" fakir hand ").

Apoplectic attacks may occur in the course of the disease, but they are not typical. Attacks of apoplexy followed by hemiplegia have sometimes led to cessation of the tremor in the paralysed limbs (Parkinson, Westphal, Bychowski), but it generally reappears. A case observed by Collet is very unusual in that the tremor suddenly ceased in one side of the body, although no marked paralysis had appeared.

The symptomatology is on the whole very uniform, the various cases showing a marked resemblance to each other. There is not the variety of type found in many other conditions, although *atypical* forms of paralysis agitans do occur. The type in which the tremor is absent or slight can, it is true, hardly be termed an unusual one, as the number of such cases is comparatively large.

The *hemiplegic* type described above is not a common variety. The cases in which the tremor or rigidity are almost exclusively limited to the legs, and thus produces an unusual condition, are very rare. Another

[1] *Arch. bohém. de Méd.*, 1902.

atypical form is that in which the muscular stiffness is absent, though the tremor is marked and extensive. I have seen cases of this kind which were difficult to diagnose from some forms of hysteria and neurasthenia, and am convinced that there is a tremor-neurosis of chronic course which can be regarded neither as paralysis agitans nor as hysteria (see my paper in the *D. m. W.*, 1905). Rabot and Béchet have described such cases. The tremor may also change its typical character and differ from the ordinary type in the great frequency of the oscillations or the increased influence of active movements.

The *attitude* may be unusual, as in rare cases (Westphal, Dutil, Oppenheim, Hansen) the head is inclined backwards, not forwards, or to one side, as in torticollis (Béchet). Atypical forms of paralysis agitans are described in the theses and papers of Gilli, Collet, and Compin, but the literature other than French has not received sufficient consideration.

The *prognosis* is favourable as to life, but there is no hope of recovery. I have found from the observation of a great number of cases that there are great differences in the severity and course of the condition; in some, there is steady progress and extension, and in others the affection hardly advances for many years, and the subjective troubles are very slight. The primary disease may be associated with symptoms of psychogenic origin which are amenable to treatment (Oppenheim, Gumpertz, etc.). French writers (Pierret, Compin) mention a rheumatic form which is said to have a comparatively good prognosis.

Differential Diagnosis.—The disease can hardly be mistaken for disseminated sclerosis. Its onset in advanced life, the character of the tremor and contracture, the absence of nystagmus, of disease of the optic nerve, and of bladder disturbances, etc., are all so characteristic that a wrong diagnosis can hardly be made in typical cases. But of late years cases have been observed in which the symptoms point convincingly to a *combination of paralysis agitans and disseminated sclerosis* (Sachs, Oppenheim-Krause,[1] Jolly [2]). This type has other characteristics, *e.g.* its onset at a comparatively early age. Tremor of an intentional character has been noted in a few cases (Gowers, Brissaud, Dejerine, Esher, Lamacq). If the illness commences with weakness and stiffness, the tremor being absent, the typical attitude of the extremities, head, and trunk will point to a definite diagnosis. Stiffness and heaviness in the limbs of one side of the body, developing in advanced life, is usually paralysis agitans, but this form is often wrongly diagnosed.

In paralytic dementia the tremor does not show the typical rhythmical oscillations, which persist during rest, and the habitual attitude of the limbs and trunk are not present, whilst the mental changes, speech disorder, and symptoms of paralysis characterise the condition. *Senile tremor* is very similar to that of paralysis agitans, but here the head is chiefly affected, the tremor is increased or brought on by active movements, and the other symptoms of paralysis agitans are absent. We do not therefore agree with Demange in classifying senile tremor with paralysis agitans. Charcot and Joffroy think that this tremor is not an attribute of old age, but has a much earlier onset and is identical with hereditary tremor—a view which I cannot accept. Raymond and Cestan would also include congenital or infantile tremor in this category. Both Charcot and Trousseau regard senile tremor as a rare symptom. In

[1] *Charité-Annalen*, xxvii. [2] *B. k. W.*, 1904.

any case our knowledge of senile tremor is very much in need of amplification. A hereditary, familial tremor, affecting mainly the head, is described by Mitchell.

I have occasionally observed a combination of paralysis agitans with senile dementia and symptoms caused by a focal disease of the brain, *e.g.* aphasia. Scherb describes this condition also. The nature of senility would lead one to expect the occasional occurrence of such combinations.

The fact that a form of spastic paralysis occurs in old age (senile paraplegia, see p. 331) is important as regards diagnosis. Senile arteriosclerosis of the brain and spinal cord may produce a condition greatly resembling paralysis agitans. In the cases of this kind which I have treated, the posture of the body, in particular, reminded me of paralysis agitans, but true paralytic conditions, *e.g.* paralysis of the bladder, dysphagia, or dysarthria, were present, or the condition exactly resembled pseudo-bulbar paralysis.[1] In another case there was anæsthesia of the spinal type, and the deep reflexes were markedly exaggerated. It is difficult to say whether these forms can in general be sharply distinguished from paralysis agitans, or whether, in accordance with a view to be discussed later, the pathological processes are identical, the differences being caused merely by their intensity and localisation.

Unilateral spastic hemiplegia, as described by Spiller (see p. 825), differs from the unilateral form of paralysis agitans in its pyramidal symptoms. The rare combination of paralysis agitans with tabes dorsalis or with tabetic symptoms, described from Oppenheim's cases by Heimann and Placzek,[2] and by Seiffer,[3] etc., may lead to difficulty in diagnosis. Salomonson's attempt to isolate this combination as a new type under a new name has found little support. Seiffer is of opinion that, in addition to the combinations, the pathological process which causes paralysis agitans may possibly, on account of its unusual localisation, give rise to these symptoms. The combination of paralysis agitans with myxœdema or myxœdematous symptoms has been described by Luzzatto[4] and Lundborg. I have once seen tremor and lateropulsion develop in a gouty patient after colchicum poisoning ; these symptoms reminded me so much of commencing paralysis agitans that I was inclined to make this diagnosis, but I found that they disappeared when the amount of colchicum was reduced. Tremor of a similar kind with recovery is said to have occurred in the course of typhoid (Clément). A similar case observed by Pennato is difficult to explain (*Rif. med.*, 1905).

Hysteria may exhibit a tremor very like that of paralysis agitans, but the oscillations are usually of greater amplitude, and their dependence upon mental processes is still more marked. Thus, it can often be stopped for a considerable time by hypnotism, and in such cases other signs of hysteria are always present.

When the symptoms of propulsion and retropulsion develop in the typical way, they may be regarded as pathognomonic. These tendencies may, however, occasionally appear in hysteria and the allied neuroses, and in rare cases of cerebellar disease. See also the observations of Petrén, Pelnar, and others, quoted on p.821.

The cases in which the symptoms *follow a trauma* are very difficult

[1] The differential diagnosis between paralysis agitans and pseudo-bulbar paralysis is also discussed by Vorkastner, *C. f. N.* 1905.

[2] *D. m. W.*, 1892. [3] *N. C.*, 1900. [4] *Riv. venet. di sc.*, 1899.

to interpret. We know that true paralysis agitans may be produced by injuries. On the other hand there is a *form of traumatic neurosis* in which the tremor and posture are the same as in paralysis agitans, but which show other symptoms not belonging to this disease (narrowing of the field of vision, disturbances of sensibility, rapid pulse, exaggeration of reflexes). These symptoms usually point to a definite diagnosis, and this is particularly important, as this form of traumatic neurosis, although very persistent, has not the progressive character of paralysis agitans.

Pathological Anatomy.—We know nothing definite as to the anatomical basis of this disease. In most cases no changes were found, and those in which a localised disease, *e.g.* a tumour in the optic thalamus (Virchow, Leyden), was present, represented a symptomatic paralysis agitans, or post-hemiplegic paralysis agitans, which should be distinguished from the true form. Recent authors (Ketscher, Borgherini, Koller, Dana, Redlich, Sander, Burzio) describe changes in the central nervous system, the spinal cord, the posterior and lateral tracts, and the grey matter, viz., sclerotic processes in the vessels and perivascular sclerosis in their neighbourhood (see Fig. 421), which involve mainly the small *vessels*, the *glia* (and connective tissue), and are identical with the well-known *senile processes* in the nervous system. Whether we are justified in regarding paralysis agitans to some extent as an *exaggerated senile degeneration* appears to me to be very doubtful. Fürstner and Erb and also Nonne have lately stated their opposition to this view. Byschowsky attributed the affection to the cerebrum. (He has subsequently described it as a functional disease of the muscles—a *myosis*.) Corresponding cell changes, such as Philipp has found with Nissl's method, and the changes in the anterior horn cells which Dana has found, and upon which he bases his audacious theory, can hardly be of any practical importance. There were no changes in the case investigated by Walbaum.[1] Carrayrou thinks a dilatation of the veins in the pons, which he found, is of some importance. Gauthier, Blocq, and others had already regarded the disease as of *myopathic* origin, but the pathological changes which would confirm this view are few and uncertain (Blocq, Sass, Dana, Schwenn [2]). Positive changes have more recently been found by Schiefferdecker-Schultze and Idelsohn. These consist partly of simple atrophy and increase of nuclei, partly of lacunar atrophy—the appearance of clear strips and spaces within the transverse section of the fibre—and in particular of changes in the muscle fibrils

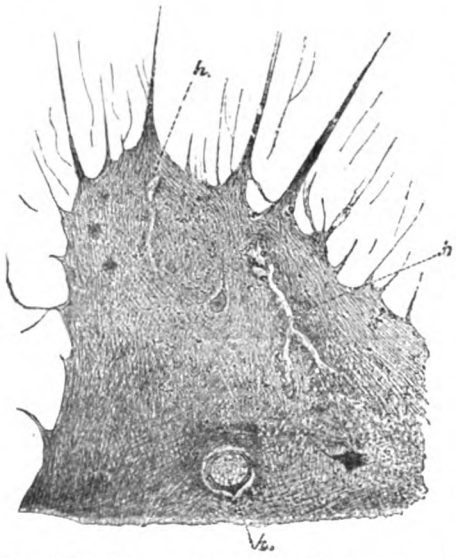

FIG. 421.—Sclerotic processes in the vessels, and perivascular sclerosis of the grey matter of the spinal cord in paralysis agitans. *h*, changes in the vessels. (Oppenheim.)

[1] *V. A.*, Bd. clxv.

[2] *A. f. kl. M.*, Bd. lxxvi.

within the spindles (the neuromuscular bundle). Schiefferdecker thinks these changes in the muscle spindles are the cause of the disease. Idelsohn emphasises the unusual prominence of the longitudinal striation of the primary fibres, etc. He has shown me his specimens, which all present these changes. But I very much doubt whether this is the causal element of the disease, as fine changes of this kind may well be of a secondary nature, *i.e.* a result of the unusual muscular activity. This point has been raised by Idelsohn himself. Changes in the muscles were also found by Camp, Catola, and Salario, whilst the investigations of Buck-Demoor and Kinichi-Naka [1] had negative results. There is no definite ground for referring the disease to the *thyroid* (Möbius, Lundborg [2]), and the parathyroid origin, assumed by some authors (Lundborg,[3] Camp, and Berkley), also seems to me very doubtful. Its nature therefore still remains quite obscure. Many writers classify it as a *neurosis*; some believe it is due to an affection of the *vascular system* of the spinal cord or to pathological changes at certain places (*e.g.* in the substantia nigra, Brissaud), and others regard it as a *disease of the muscles*. This is opposed by the theory of a disturbance of metabolism due to senile degeneration of the thyroids or parathyroids, and finally by the teaching that paralysis agitans is merely a *syndrome*, which may be produced in numerous ways (Teissier, etc.).

Treatment of this disease is not very satisfactory, but it may do much to alleviate the symptoms and mitigate the severity of the disease.

In the first place everything which excites the patient's mind should be avoided. He should be withdrawn from the society of others, and should as far as possible live an isolated life, seeing only those to whom he is accustomed, who will not excite him, and who thoroughly understand his condition. The requirements and experience of each patient should of course be fully taken into consideration. Fashionable health resorts are not suitable; but residence in the *country*, the *hills* or *woods*, may be very helpful, provided the patient can live quietly there.

All energetic methods of treatment should be avoided. Energetic cold-water treatment is therefore unsuitable, whilst rubbing for a short time with warm water, warm full baths, or even cold half-baths may have a good effect.

Electrotherapy is of little service, but I have seen recently some, or even a marked degree of improvement in several cases from the use of electric baths, especially the bipolar faradic and the four-cell baths. Gentle passive movements may soothe the tremor. I have often used and recommended this method, in which the movements must be varied so as not to produce the tremor; and while it does not cause any permanent improvement, it has in many cases a beneficial influence which may last for several hours. I have not found massage of any service, but it is recommended by Hoffa and others. J. M. Taylor, Friedländer, and others have been successful with gymnastic exercises, but I should advise the avoidance of any over-strain in this condition.

Nerve-stretching and the use of *suspension* can no longer be considered practicable.

As regards drugs, strong purgatives and diaphoretics should be carefully avoided. I have done harm in one case by prescribing a *preparation of salicylate*, and in another *Dover's powder*. *Hyperidrosis*, which persisted and was very troublesome, commenced from that time. There

[1] *A. f. P.*, Bd. xli. [2] *Z. f. N.*, xix. [3] *Z. f. N.*, xxvii.

need be no hesitation in prescribing *bromides*, which may alleviate both the restlessness and the feeling of anxiety. *Tincture of veratrum viride*, in doses of three to four drops several times a day, has lessened the intensity of the tremor in a few cases. I can recommend the careful use of this drug and also of tincture of gelsemium. Erb believes in arsenic. He gives Fowler's solution, aqua fœniculi, and tinct. nux vom., in equal parts, six to twelve drops being given as a dose. In severe cases with violent tremor which prevents rest at night, *morphia* must be resorted to. Subcutaneous injections of morphia procure many hours' rest, but of course its ill-effects have to be also accepted. *Atropin* is also used. *Hyoscin* (scopolamin. hydrobrom.), which Erb recommends, and *sulphate of duboisin*, which has more recently been used by Mendel, Francotte, etc., are effective palliative drugs, and may still the tremor for some hours. Both drugs are very poisonous, and must be given in doses which will stop the tremor without causing symptoms of intoxication (vertigo, headache, visual disturbance, nausea, dryness of the throat, etc.). Hyoscin should be given in doses of two to four decimilligrams once or twice a day, and duboisin in the same dose (Erb thinks a little more is required). These are best given subcutaneously, but they may also be effectual given internally. Erb strongly advocates hyoscin or scopolamin hydrobrom., and I am able to state that I have found it exceedingly helpful in many cases. Hilbert, Higier, and others have used it for a whole year without any ill effect, and I have also found this to be so, although I have more often had to interrupt the treatment on account of symptoms of poisoning. It is very important that the solution should be fresh and the apparatus clean. Hilbert advises that the subcutaneous injection should only be given every second or third day, the drug being used internally in the intervals. Rosenfeld, Bumke, and others have found that the dose could be increased without danger. Marie and Roussy also give up to two mg. ($\frac{1}{33}$ grain) a day. Some of my patients are only benefited by subcutaneous injections, and others find internal administration of the drug almost as helpful. I know one case in which the tremor entirely ceases for several days after scopolamin poisoning. I have found duboisin to have a palliative effect in a few cases, but in others its use was associated with marked loss of strength. In one case in which duboisin had proved quite ineffectual, the administration of two and a half decimilligrams of hyoscin once a day produced an extraordinary remission, with return of sleep, increase of strength, and a feeling of complete health. Hart also reports favourable results. Arsenic, whether given subcutaneously or internally, is of little value.

Podack has recently recommended rhizoma scopoliæ carniol. (in tabloids of 3-6 grs.), of which Kétly also approves (*Therap. d. Geg.*, 1903).

Thyroidin, which Lundborg prescribed on theoretic grounds, had no effect. Parathyroid preparations have lately come much into use, and Berkley[1] reports good results from them. I used the drug for several years, in the preparation advised by Freund and Redlich, but with no definite result. Parhon-Urechié (*R. n.*, 1907) report benefit from preparations of the pituitary body.

The most important point in the later stages is to procure a *comfortable position* for the patient, and to contrive arrangements which will

[1] *Med. News*, 1905.

enable him to change it as far as possible. The bedclothes should not be too tightly arranged or too heavy during his sleep. His nourishment should be strengthening and non-irritating. Alcohol should be given only in small quantities, but its effect should be judged in each case. Straining at stool should be avoided. Movement in the open air is good or even necessary for many cases, but over-fatigue must be urgently forbidden. One of my patients was undoubtedly made worse by an expedition into the hills.

Charcot pointed out that persons with paralysis agitans usually feel better driving in a carriage and especially in a railway train. This observation—which is not of general application—has led to the adoption of mechanical treatment, the patient sitting in a chair which is constantly rocking (fauteil trépidant), but I have not found it of any practical benefit.

The value of psychotherapy in this disease should not be overlooked.

Diseases of the Sympathetic Nervous System

Angioneuroses and Trophoneuroses

Anatomy and Physiology

Bibliography in S. Mayer, "Das sympath. Nervensystem"; Hermann's "Handbuch der Physiologie"; Schultz, "Das sympath. Nervensystem": Nagel's "Handbuch d. Physiol. des Menchen," 1906; Lewandowsky, "Die Funktionen des zentralen Nervensystems," Jena, 1907; Langley, papers in *Philos. Trans.* and *Journ. of Physiol.*; Ascher-Spiros, "Ergebnisse d. Physiol.," ii. 2; Grützner, "Vasomot. Nerven," abs. *A. f. P.*, Bd. xlii.; Cassirer, "Die vasomotorisch-trophischen Neurosen," Berlin, 1901; Herz, "Zur Lehre von den Neurosen des periph. Kreislaufapparates."

The *sympathetic* nervous system—the gangliated nerves of Kölliker—consists (1) of the *funiculus marginalis, i.e.* a chain of ganglia lying along each side of the spinal column, which are united by longitudinal fibres into a tract which extends from the head to the coccyx; (2) of *rami communicantes, i.e.* fibres which arise mostly from the spinal cord, leaving it with the roots, and passing from these or from the spinal nerves which they form, into the ganglia or the funiculus marginalis. The white or efferent rami communicantes are contained chiefly or entirely in the *anterior* roots, but vasodilator impulses are also said to be conveyed from the spinal cord towards the periphery in the tract of the posterior roots (Stricker, Bonuzzi, Kohnstamm, Bayliss), a view thought by Langley and Lewandowsky to be very doubtful, but maintained by Grützner. The rami communicantes also contain fibres coming from the sympathetic and entering into the tract of the cerebro-spinal nerves; (3) of peripheral branches which leave the funiculus marginalis at every level and pass to the viscera, blood-vessels, glands, etc., that is to say to the smooth muscles. They anastomose at many points with the branches of the cerebro-spinal nerves, especially of the vagus system and the sacral nerves, and thus form the sympathetic plexus, which contains peripheral ganglia at many points.

The sympathetic is divided into portions for the head, neck, thorax, abdomen, and pelvis.

The cervical sympathetic occupies a special position, as above the inferior cervical ganglion it ceases to receive white rami communicantes from the spinal cord. The spinal branches for the cervical ganglia arise more from the cord, pass into the inferior cervical or stellate ganglion, and through this into the cervical sympathetic. The latter therefore forms so to say the white rami communicantes for the middle, and especially for the upper cervical ganglion. In man, however, the cervical sympathetic often receives a small accession from the last cervical nerves.

Langley, whose researches were mainly on the cat, found, as Gaskell had already done, similar conditions in the sacral sympathetic, the rami communicantes of which emerge from the spinal cord by means of dorsal and especially of lumbar roots. The spinal sympathetic therefore has its origin chiefly between the cervical and upper lumbar regions. In man the second or third lumbar nerve probably forms the lowest limit; the nerves below these do not probably give off any white rami communicantes. (The connection of the sympathetic with the grey rami is everywhere

present.) The sacral cord may be so far included in the area of origin of the sympathetic, as the nervus erigens has its origin at the level of the second and third, or the second to fourth sacral segments.

For the earlier terms of vegetative, visceral, etc., Langley (*Br.*, 1903) has substituted that of *autonomic* nervous system. In addition to the sympathetic in the strict sense, *i.e.* the marginal column which has its origin in the dorsal and superior lumbar cord, he distinguishes a bulbar autonomic and a sacral autonomic system. The former includes the vagus (or vagus, glosso-pharyngeal, and intermedius) in so far as it extends to organs and structures which are not under voluntary control. The sacral autonomic region includes the nervus erigens. From very careful experimental studies he has drawn precise data as to the sphere of innervation of the various segments of the sympathetic or autonomic nervous system, but the results of experiments on animals cannot be directly applied to man, although his latest publications ("Ergeb. d. Physiol.") refer entirely to man. This also applies to the experimental observations of Laignel-Lavastine,[1] referring chiefly to the solar plexus.

Medullated fibres pass through the white rami communicantes to the sympathetic tract, whilst the nerves arising from its ganglia are non-medullated. According to Kölliker some fine medullated fibres also arise from the cells of the sympathetic ganglia, *e.g.* the ciliary nerves. The nerve appears white or grey according to which of these fibres predominate. The splanchnic nerve, for instance, contains a large number of cerebro-spinal fibres, which give it a white appearance.

As regards the *course of the fibres* in the sympathetic nerve, we may assume the following conclusions from the investigations of Lenhossek, Gaskell, Retzius, Gehuchten, Langley, Kölliker, Edgeworth, Morat, Onuf-Collins,[2] and others:

The efferent fibres which run in the white rami communicantes, and arise from the spinal cord, enter as *pre-cellular* fibres into the sympathetic ganglia, and surround its cells with terminal baskets. Here therefore ends the neurone of the first order. The ganglion cells of the sympathetic ganglia now send out the *post-cellular* (post-ganglionic) fibres, which pass mainly towards the periphery and terminate in the smooth muscles, etc. These with their cells of origin form the neurone of the second order. But all the precellular fibres do not directly terminate in the ganglion with which the ramus communicans is connected; some of them pass through several ganglia or go to the peripheral ganglia of the sympathetic plexus (Langley). This is the case, *e.g.* with the splanchnics. Langley thinks lateral branches also end in the ganglia which are traversed by the precellular fibres, so that a pre-ganglionic fibre may enter into connection with several sympathetic ganglia. The post-ganglionic branches enter partly into the spinal nerves and through these pass to the periphery, or go as offshoots of the sympathetic ganglia with the arteries to the viscera. This is chiefly the case as regards the post-ganglionic fibres of the sympathetic ganglia contained in the abdominal and pelvic cavities (cœliac, mesenteric, etc.).

The *physiology* of the sympathetic has been greatly advanced during recent years by the investigations of Anderson, Gaskell,[3] Langley, Lewaschew,[4] Abadie, Lapinsky,[5] Morat, Onuf-Collins, etc. The surgery of the sympathetic, in particular the cases of Chipault, Jonnesco,[6] Abadie, Jaboulay, etc., has also contributed to our knowledge of the functions of this nerve, but many points are still obscure.

The sympathetic nerve extends into every part of the body which contains smooth muscles, especially into the vessels, stomach, intestine, trachea, bronchi, lungs, urethra, bladder, and uterus. It also innervates the dilator pupillæ muscles, the smooth muscles of the eyelids, the orbitalis, and the erectores pilorum, as well as the salivary and sweat glands, the glands of the gastro-intestinal tract, the œsophagus, and the heart. It is not, however, the only nerve which innervates these structures, sharing this function to a great extent with the vagus.

Its innervating influence upon the vessels is not limited to those with non-striped muscles. The experiments of Rouget, S. Mayer, and especially those of Steinach and Kahn[7] seem to show that the capillary walls also possess the power of contractility by means of cells which are

[1] *Arch. gén. de Méd.*, 1903; *Gaz. des. hôp.*, 1903; *Thèse de Paris*, 1903; *Journ. de Neurol.*, 1904, etc.

[2] *Journ. of Neurol.*, 1898; *Arch. of Neurol.*, 1900.

[3] *Journ. of Physiol.*, vii., x., etc.

[4] *V. A.*, Bd. xcii.; *Pflügers Arch.*, 1882.

[5] Dubois, "Arch.," 1898; *Z. f. N.*, xvi.; *V. A.*, Bd. clxxxiii., etc.

[6] "Chirurgie du grand Sympath.," Congrès internat. de Lisbonne, 1906; ref. *R. n.*, 1906.

[7] *Pflügers Arch.*, Bd. xcvii.

analogous with the cells of non-striped muscles. According to Goltz, nerve fibres also extend to the veins, and Grützner has lately specially maintained the independent action of the vessel walls and the veins, under the influence of their nerves.

The bulbar autonomic system has a great share in the innervation of the heart, gastro-intestinal tract, trachea, and lungs. According to Langley the vagus conveys inhibiting impulses to the heart, and both exciting and inhibiting impulses to the gastro-intestinal canal, whilst the sympathetic contains the *accelerators* for the heart and the *constrictors* for the blood-vessels. He ascribed to the sacral autonomic system a vaso-dilator influence upon the arteries of the rectum, anus, and the external genital organs, and an exciting (and partly inhibiting) influence upon the smooth muscles of the colon, rectum, anus, and bladder, whilst the sympathetic nervous system conveys on the one hand vaso-constrictor impulses, and on the other shares with the sacral autonomic system the innervation of the smooth muscles of the descending colon, rectum, anus, urethra, bladder, and genitals. To the latter it conveys chiefly inhibiting impulses. It must always be borne in mind, however, that these assumptions rest partly on the uncertain results of animal experiments. It has been specially shown by Pawlow [1] that the vagus and sympathetic contain secretory fibres for the gastric glands and the pancreas.

The "pilomotor" fibres, the functions and distribution of which have been studied with special care by Langley, are less important in man. He has established close relations between the vasomotor and pilomotor innervation of the skin, and has made important statements as to the area of innervation of the various segments of the spinal cord and sympathetic ganglia, which we cannot here consider. Durdufi, Fränkel, Thoma, Lapinsky, and others have shown by experiment the *trophic* value of the sympathetic. The former saw section of the cervical part of this nerve in young animals followed by hypertrophy of the ear, and Lapinsky in this way produced changes in the vessel wall. Jores,[2] who obtained no results with this experiment, is opposed to Lapinsky's views.

This is not the place to discuss the bearing of these alterations upon the vasomotor function of the sympathetic nervous system (see p. 66). It has been long since shown by clinical observations that disturbances of vasomotor innervation lead in the end to degenerative changes in the vessel walls, a fact which has been emphasised by Oppenheim (see p. 1136), Romberg, Herz, and others. Onuf and Collins found severe *trophic* disturbances, especially in the skin, in cats after excision of the stellate ganglion and the superior thoracic sympathetic. As regards the experimental production of and the theories upon alopecia, we would refer to p. 67.

Jonnesco, from his numerous experiments by section and removal of the ganglia in man and in animals, concludes as follows with regard to the parts innervated by the cervical sympathetic: It contains (1) dilator fibres for the pupils; (2) motor fibres for the non-striped muscles of the orbit and upper eyelid; (3) vasoconstrictor fibres for the mucous membrane of the cheeks, lips, gums, face, tongue, etc., and for the brain; (5) secretory fibres for the salivary, tear, and sweat glands of the head; (6) accelerating fibres for the heart; (7) inhibiting fibres for the heart; (8) respiratory fibres, etc.

There are various contradictions between the statements of different authors. Langley maintains that the blood-vessels of the central nervous system receive little or no innervation from the sympathetic. According to Morat the cervical sympathetic contains a system of partly antagonistic fibres, so that its stimulation may have different results. Jonnesco thinks the vaso-dilators react to weak, and the vasoconstrictors to strong stimuli. Langendorff (*Kl. Mon. f. Aug.*, Bd. xxxviii.) found differences in the condition of the pupils according to whether he resected or excised the sympathetic below the superior cervical ganglion. Chipault, in his operations on the various segments of the cervical sympathetic, has also found differences in the condition of the pupils, etc. Onuf and Collins think that (in the cat) the cervical sympathetic contains not only fibres for dilatation, but also those for constriction of the iris, but this is contradicted by Langley's statements. Lewandowsky may also be consulted on this subject.

The upper extremities receive their sympathetic (or "autonomic") nerves through the roots of the fourth to the tenth dorsal nerves, which send their rami communicantes through the funiculus marginalis and the stellate ganglion to the brachial plexus. Onuf and Collins think that the sudoral fibres of the fore-limbs do not all go through the stellate ganglion. The view of Claude Bernhard, that the sympathetic fibres for the arm pass from the middle dorsal cord directly through the sympathetic to the vessels of the arm, without passing through the brachial plexus,

[1] "Die Arbeit der Verdaungsdrüsen," Wiesbaden, 1898.

[2] *Zieglers Beiträge*, Bd., xxxii. Compare with Lapinsky, *V. A.*, Bd. clxxxiii.

is opposed by Egger on the ground of his clinical observations. According to Langley, the preganglionic tracts of the fore-limbs end in the stellate ganglion.

Spallita and Consiglio [1] have made some statements as to the origin of the vasomotors of the sciatic, crural, etc. Langley, Onuf-Collins, and Lapinsky have also pointed out that our knowledge of the sympathetic innervation of the lower limbs in man is still very unsatisfactory.

Investigations into the sympathetic innervation of the abdominal viscera and its site of origin in the medulla have been made by François Franck and Hallion,[2] Head, Langley, Onuf-Collins, Laignel-Lavastine, etc. As regards Head's statements, which bear specially on so-called reflex pain in the region innervated by the corresponding spinal nerves, see p. 108. See also Rynberk, *Arch. di Fisiol.*, 1907. According to François Franck and Hallion the liver is supplied from the sixth dorsal to the second lumbar nerves. We need not here consider the details given by Onuf-Collins and Laignel-Lavastine, nor the theory of the chromaffine cell-groups of the sympathetic and their relations to the suprarenals.

According to Kölliker, the sympathetic does not itself contain sensory elements; it is merely traversed by sensory fibres, which come from the viscera and reach the spinal cord in its tract by means of the rami communicantes of the posterior roots. Kölliker thinks they arise from the spinal ganglia, Onuf-Collins from the sympathetic ganglia. The latter think they make their way into Clarke's column and the intermediary grey matter. The efferent fibres of the sympathetic arise chiefly from the cells of the grey matter, the so-called paracentral nuclei and lateral horn; Langley states that they come from the lateral column cells. Gaskell and Sherrington also believe that the lateral horn is related to the sympathetic. See the paper by L. Jacobsohn (*N. C.*, 1908). Many writers (Marinesco, Kohnstamm, Hunt, Winkler, Onuf-Collins) assume that the so-called dorsal vagus nucleus plays a similar rôle.

The sensory tracts chiefly convey the sensations arising from the viscera. Under normal conditions these, as we know, are quite unimportant: see, however, Lennander (*Mitt. aus Grenzg.*, xv., xvi.; *Z. f. Chir.*, Bd. lxxiii.); Kast-Meltzer (*B. k. W.*, 1907), and L. R. Müller (*Mitt. aus Grenzg.*, xviii.). They also serve for conduction of centripetal, unconscious impulses, and of reflex processes to the sympathetic. According to François-Franck and others, similar reflex processes take place in the sympathetic ganglia themselves. Edinger is also of opinion that, according to the fulness and distension of the vessel walls, sensory excitations arise from these, and by stimulation of the sympathetic ganglia produce reflex muscular contractions in the vessel wall itself. Kölliker does not agree with this. Symptoms which may be regarded as reflex have also been experimentally produced by Claude Bernard, Langley-Anderson (*Journ. of Physiol.*, xiii.), Pawlow, and Bayliss-Starling (*Journ. of Physiol.*, xxvi.).

Here we are dealing chiefly with secretory and motor processes which may be elicited in that part of the sympathetic which is distinct from the cerebro-spinal nervous system. According to Langley they should be regarded not as true reflexes, but as preganglionic axone-reflexes; he attributes these to the fact that the preganglionic fibres give off lateral branches, so that in stimulation of their central stump the excitation is conveyed by the lateral branch to the peripheral end organ. This explanation has a somewhat artificial air, and many facts go to confirm the occurrence of reflex movements in the sympathetic. Langley has not of late been so much opposed to this view. Clinical observations of this kind have been described by Buch, and some other data have been given by L. R. Müller (*A. f. kl. M.*, 1889).

Nottebaum ("Inaug.-Dissert.," Marburg, 1897), after section of the cervical sympathetic, failed to find secondary degeneration in the spinal cord and oblongata. Lapinsky and Cassirer (*Z. f. N.*, xix.)—in opposition to Biedl, Hoeben, Huet, Collins, Onuf, Anderson, Herring, and others—had negative results from their experiments, as removal of the cervical sympathetic ganglia in guinea-pigs was followed by no pathological change, either in the fibres or cells of the corresponding parts of the spinal cord.

The symptoms of lesion of the sympathetic have been specially studied with regard to the cervical sympathetic, partly by means of experiment on animals (Cl. Bernard) and on decapitated persons (R. Wagner, H. Müller, G. Fischer), and partly in cases in which injuries have involved the sympathetic. In France especially, surgical resection of the cervical sympathetic and its ganglia, for the cure of epilepsy, exophthalmic goitre,

[1] *Arch. ital. de Biol.*, xxviii., and abs. *Jahresber. f. Neurol. u. P.*, 1898.
[2] *Arch. de Physiol.*, 1896 and 1897.

and glaucoma, has of late years enabled us to study its secondary results in man (Chipault,[1] Jonnesco,[2] Abadie, Laborde,[3] Braun, Jaboulay, etc.).

Section of the sympathetic at the neck causes, according to the well-known investigations of Cl. Bernard, dilatation of the blood-vessels on the corresponding side of the head, increase of the temperature of the skin, contraction of the pupil and palpebral aperture of the same side, and sometimes retraction of the eye-ball (enophthalmus). This vascular dilatation may extend to the tympanic cavity, the choroid and retina, and to the meninges of the brain.

Stimulation of the cervical sympathetic produces contraction of the vessels, fall of temperature, dilatation of the pupil and palpebral aperture, protrusion of the eyeball, and sweating on the same side of the head. It may also cause dilatation of the vessels in the face (Morat, Abadie). The secretion of the parotid gland and the salivary glands in the lower jaw has also been stimulated, and the action of the heart accelerated from the cervical sympathetic.

François Franck regards some of these symptoms and others which appear on the contralateral side as reflex, due to stimulation of sensory fibres contained in the cervical sympathetic. He doubts whether the sympathetic contains vaso-dilator fibres for the brain. The statements of different authors as to the relations of the cervical or thoracic sympathetic to the thyroid and the heart are by no means unanimous. Thus Abadie attributes the vasodilators of the thyroid gland to the sympathetic, whilst Cyon thinks they belong to the depressor. Köster (Pflüger's "Arch.," Bd. xciii.) regards the latter as the sensory nerve of the aorta, and thinks it arises from the jugular ganglion. According to Franck the vasoconstrictors of the thyroid alone run in the cervical sympathetic, the vaso-dilators being contained in the superior laryngeal. He thinks it has also only a vasoconstrictor influence upon the brain, so that its section would cause cerebral hyperæmia.

Disturbances corresponding to experimental section have sometimes been observed in man after stabbing, shooting, or cutting injuries, operations on the neck, or direct resection and excision of the sympathetic or its ganglia (Chipault, Jonnesco, Jaboulay, etc.), or after lesion of the nerve by tumour-compression (glandular tumours, struma, aortic aneurism, etc.) (Ogle, Nicati,[4] Seeligmüller,[5] Möbius, Stewart,[6] etc.). Goitres do not need to be very large to compress the sympathetic. It is most easily affected by flat goitres which extend more or less far back (Heiligenthal [7]). He also points out that the cervical vessels and the vagus can make way for the intruding tumour, whilst the sympathetic is firmly fixed. In a case described by Holz, acute swelling of the thyroid first produced symptoms of irritation, then of paralysis of the sympathetic. Observations by Horner,[8] and Michel [9] point also to a *puerperal* origin of sympathetic paralysis, and disease of the sympathetic has been observed in diabetes. Some cases seem to show that tubercular processes of the apex of the lung may directly involve the sympathetic (Bouveyron,[10] Barrel, Souques [11]). Addison's disease, exophthalmic goitre, facial hemiatrophy, and lately also glaucoma have specially been attributed to disease of the

[1] *R. n.*, 1898, 1899, *et seq.*
[2] *Arch. des Sciences méd. de Boucarest*, 1900 ; also "Chirurgie du grand Sympathetique," Congrès internat. de Lisbonne, abs. *R. n.*, 1906.
[3] *R. n.*, 1898.
[4] "La paralysie du nerf sympath. cervical.," Diss. Zürich, 1873.
[5] *B. k. W.*, 1870, 1872, and "Habilitationsschrift," Halle, 1876.
[6] *Brit. Med. Journ.*, 1901.
[7] *A. f. P.*, xxxiii.
[8] *Kl. M. f. Aug.*, vii.
[9] *Graefe-Saemischs Handbuch*, iv.
[10] *Lyon méd.*, 1899, and *R. n.*, 1902.
[11] *Soc. méd. des hôp de Paris*, 1902.

sympathetic. Finally, I have observed a case of *hereditary sympathetic paralysis*[1] (in a twin in whom some or all of the symptoms developed in later life), and have brought it into analogy with certain forms of hereditary paralysis of the ocular muscles. The combination of a congenital, unilateral disturbance of innervation of the cervical sympathetic with congenital ocular paralysis has recently been described by Michel.[2] I have seen another case in which only some of the symptoms were present in early childhood, recurring afresh in adult life and showing the complete picture of unilateral paralysis of the cervical sympathetic. I have also stated that a *congenital weakness* of the sympathetic (especially the cervical) may cause it to be less resistant to such harmful influences as compression and trauma.

FIG. 422.—Narrowing of the left palpebral aperture; diminution of the left pupil, and slight retraction of the left eyeball in compression of the left sympathetic by a left-sided goitre. (Oppenheim.)

Negro has recently noted that the syndrome of Claude-Bernard and Horner is a common stigma of degeneration, especially in epileptics.

The following are the symptoms of *paralysis* of the cervical sympathetic: (1) Contraction of the pupil of the same side (the most common symptom), the light reflex being present but sometimes sluggish and slight; (2) Contraction of the palpebral fissure of the same side (Fig. 422); (3) Retraction of the eye-ball, which is less constant and of gradual development. It has been ascribed to atrophy of the fat of the orbit, and to paralysis of the so-called orbital muscle, a smooth muscle which is but slightly developed in man (Nicati). Softening of the eyeball has occasionally been noted. (4) Dilatation of the vessels on the same side of the face and head. This symptom is not only often absent, but the vessels have even been observed to be contracted under similar circumstances. Nicati's view that this represents a second stage, following the dilatation, has rightly been rejected, although similar opinions had already been expressed by physiologists and had led to the assumption that the sympathetic ganglia in the vessel walls made their regulating influence felt in course of time (Goltz[3] and Ostrumoff[4]). It may be taken for granted that the vascular constriction only occurs in lesions which cause merely partial interruption of conduction, so that irritative and paralytic symptoms may be combined. Heiligenthal states that the condition of the temperature of the skin and the vascular distension are in themselves less characteristic than the kind of reaction to the irritation affecting the vascular nervous system—mental excitement, physical fatigue, etc. This has been confirmed by Conzen.[5] (5) Anidrosis of the same side of the face. This is an inconstant or temporary symptom (Jonnesco-Floresco[6]). Hyperidrosis has occasionally been present under similar conditions, and is usually a transient symptom in surgical paralysis of the sympathetic. Jendrassik[7] states that he has observed anidrosis

[1] *N. C.*, 1903.
[2] *Z. f. Aug.*, x.
[3] *Pflüger's Arch.*, xi.
[4] *Pflüger's Arch.*, xii.
[5] "Inaug.-Diss.," Leipzig, 1904.
[6] *Journ. de Physiol.*, iv.; and *Arch. méd. de Boucarest.*
[7] *V. A.*, Bd. cxlv.

in lesions situated high up in the neck, and hyperidrosis in lower lesions. (6) In rare cases emaciation of the same side of the face (Seeligmüller, Möbius, Oppenheim, Jaquet, Bouveyron, Barrel; compare the chapter on facial hemiatrophy). Heiligenthal thinks that this symptom is due to atrophy of the fatty tissue.

Premature greyness of the hair on the affected side of the head has been observed in rare cases (Oppenheim). Alterations of the cardiac functions have not been noted in man under such conditions. Possibly the sympathetic of the opposite side suffices to regulate the action of the heart (Nicati), or it may be innervated by much lower segments of the sympathetic. (According to Langley these fibres terminate in the stellate ganglion.) Möbius points out that sensory stimulation on the side of the paralysis may not cause the pupils to dilate, but later investigations have had other results (see p. 83).

The symptoms produced by surgical paralysis of the cervical sympathetic due to resection of the nerve or the highest ganglion are the same as those just described. The oculo-pupillary symptoms are most constant; hyperæmia and rise of temperature are usually present, but these symptoms and the hyperidrosis or anidrosis usually disappear within one or more days. Hemiatrophy has also been said to follow this operation. Donath found the resistance of the skin to electricity diminished on the corresponding side of the face. Jonnesco includes increased salivary, nasal, and lachrymal secretion among the transient symptoms. Heiligenthal also noted lachrymation in his case.

In bilateral resection the oculo-pupillary symptoms are not always the same in both eyes. We cannot here discuss the experimental studies bearing upon paradoxical dilatation of the pupils (Langendorff, Anderson, Langley), nor the theory of the automatism of non-striped muscles (Lewandowsky [1]).

Our knowledge of the symptoms of *irritation of the sympathetic* in man, is still more uncertain, but it should be remembered that the lesions which produce it almost always give rise at the same time to the conditions necessary for paralysis of the sympathetic fibres. The symptoms of paralysis usually follow or occur simultaneously with those of irritation. Seeligmüller has observed dilatation of the pupils along with flattening of the cheeks. Dilatation of the palpebral fissure and protrusion of the eyeball are also found. F. Pick noted sluggish reaction of the dilated pupil. Conzen states that irritation of the sympathetic gives rise to hyperidrosis, but this by no means follows as a matter of course.

We know little with regard to *pain* in the parts supplied by the cervical sympathetic due to lesion of that nerve, but from personal experience and an observation by Bouveyron, I suspect that there may be neuralgia in the affected area. Kocher makes the interesting statement that pain in the ears, jaws, etc., may follow operation, and especially traction upon the cervical sympathetic.

As regards differential diagnosis, we should remember that diseases of the spinal cord (ciliospinal centre) may produce some of the symptoms just described, that many of them may arise from the cerebral cortex, and that some of them occur in the functional neuroses. It should be

[1] *Ber. d. Berl. Akad.*, 1900. See also "Die Funkt. d. zentral Nerv.," Jena, 1907.

specially borne in mind that some of the phenomena, dilatation of the pupils in particular, may very often be of a reflex nature.

We have little definite knowledge regarding *diseases of the thoracic and abdominal sympathetic*. The relations of the highest thoracic sympathetic to the thyroid gland and the heart have already been referred to. It is also certain that it contains the vasomotors for the vessels of the viscera, which for the most part run in the *splanchnic*, that the movements of the intestine, uterus, bladder, etc., are mostly under the control of the sympathetic, and that it contains motor and inhibitory fibres for the muscles of the abdominal organs. Experimental section or resection of the splanchnics is said not to produce permanent disturbance of the functions of the intestine and kidneys (Vogt-Popielski).

The *neuralgia* which is localised in the viscera has been attributed to an affection of the cœliac plexus (Anstie, Fürbringer, Buch, Hoffmann, Robinson, etc.). Buch [1] goes furthest in this respect. He states that in healthy persons the sympathetic, with the exception of the splanchnic, is completely insensitive, whilst in inflammatory and other changes the sympathetic nervous system becomes the starting-point of acute pain, in the form of gastralgia, hepatic neuralgia, etc., and of corresponding tenderness to pressure. Secretory disturbances of various kinds may also have a reflex origin. F. A. Hoffmann [2] attributes the following syndrome to the cœliac plexus : Pain in the upper abdominal region, radiating into the sacral and gluteal regions, polyuria, constipation, and stools resembling scybala. I have seen colourless or greyish-white stools as a transient symptom in severe attacks of pain of this kind in the course of a climacteric neurosis.

The experimental results of Onuf and Collins would lead one to expect disturbances of digestion, impairment of the general nutrition, trophic changes in the skin, etc. According to their investigations, almost all the secretory glands are influenced by the sympathetic. Laignel-Lavastine observed hyperæmia of the abdominal organs, vomiting, diarrhœa, oliguria, tachycardia, general loss of strength, etc., after removal of the solar plexus. Jaundice brought on by emotion has also been noted (Frerichs, Potain, Picard, Reinboldt).

Diseases of the sympathetic (degeneration) have been often observed in *Addison's disease*. The theory that Addison's disease is due to degeneration of the chromophile cell-groups of the sympathetic requires further study.

French writers have lately spoken of surgical treatment of the abdominal and pelvic sympathetic (Jaboulay, Patel-Viannay,[3] Vallas-Cotte [4]), and have stretched the splanchnic in gastric crises, apparently with success. We may at least expect that this treatment will advance our knowledge of the functions of the abdominal sympathetic.

In a very obscure case of *abscess-formation* along the thoracic portion of the spine with atrophy of the sympathetic, a *unilateral œdema* developed during life and spread over the whole side of the body. Swelling of the liver and ascites have in one case been ascribed to the splanchnic nerve, but such cases are very rare and difficult to explain.

[1] *W. kl. R.*, 1902 ; and Dubois-Reymond's *Arch.*, 1901. See also Hönck, *W. kl. R.*, 1901, and *C. f. N.*, 1907 (abs.).

[2] *M. m. W.*, 1902. [3] *Gaz. des hôp.*, 1904. [4] *Lyon méd.*, 1906.

The pathology and pathological anatomy of the sympathetic are still very obscure. In the earlier literature, which has been collected by Eulenburg and Guttmann,[1] atrophy and degeneration are frequently mentioned. Fleiner, Schapiro, Hezel, Marchand, Jellinek, and others subsequently reported positive changes, and Gaupner has since then reviewed his own cases and those published by other observers. See also Wiesel (*Z. f. Heilk.*, xxiv.) and Neusser (*Nothnagels Handbuch*, xviii.). But from all the published literature we cannot definitely ascertain the pathology of the sympathetic, nor can we say of any single, precisely defined morbid process in the sympathetic that it corresponds to a characteristic clinical condition. Some of the changes found have also been described in general marasmus and in diseases of the central nervous system (tabes, combined column diseases, disseminated sclerosis (Graupner)), and most frequently, though not always, in Addison's disease and exophthalmic goitre. Ehrich (*Bruns Beitr.*, xxviii.) has found certain alterations in the cervical sympathetic excised from a patient with exophthalmic goitre, but he regards these as secondary. The work of Langley and of Onuf and Collins seems calculated to throw more light upon this obscure subject.

Angioneuroses (and Trophoneuroses)

Many of the symptoms arising from the sympathetic nervous system have already been discussed, especially in the chapters on hysteria and neurasthenia. The part played by the sympathetic system in emotional processes has been considered there, and its great importance as regards the psychopathology of hysteria and neurasthenia has been ascribed to this relation. Although they represent only one component of the syndrome, there are, as already said, forms and cases of neurasthenia in which the symptoms relating to the vasomotor or sympathetic nervous system are the chief or only manifestation of the disease.

These include sudden attacks of flushing in the face, head, and neck, accompanied by a feeling of heat, pulsation, and sometimes by hyperidrosis. The sufferer complains of palpitation, rush of blood to the head, noises in the ears, a mist before the eyes, fear, etc. Consciousness is usually retained. Although this syndrome, which in its chief symptoms indicates a transient *paralysis of the vasomotors* (or irritation of the vasodilators) of the head, usually occurs along with hysteria and specially with *neurasthenia*, it may attain a certain independence (see chapter on cerebral hyperæmia) and appear in otherwise healthy persons. Masturbation and sexual excess are occasional causes.

There are also *family, hereditary* forms of this kind, and in some cases the involvement of the sympathetic nervous system may be the main or the only indication of a *hereditary* neuropathic diathesis. I have already pointed out, on the ground of observations by Michel and myself, that it may manifest itself as *congenital paralysis* or weakness of a certain section of the sympathetic, namely the cervical. The hereditary nature of migraine and the family occurrence of certain forms of vasomotor neurosis (Fürstner,[2] Bruns, Diehl, Meige, F. Mendel,[3] and others) are related to these facts.

The sympathetic system is also markedly involved in the symptoms of the *climacteric*, which is not surprising in view of its intimate relations to menstruation. Congestion, vertigo, tachycardia, universal and circumscribed hyperidrosis, angina pectoris, gastric disorders, clinical conditions resembling exophthalmic goitre[4] (*q.v.*), etc., also belong to this class;

[1] "Die Pathologie des Sympathicus," Berlin, 1873.

[2] *Mitt. aus Grenzgeb.*, xi.

[3] *B. k. W.*, 1902.

[4] It is said that such conditions may be artificially produced by castration (Dalché, abs. *C. f. Gr.*, 1902; Matthieu and Jayle).

and I have occasionally found oculo-pupillary symptoms (narrowing of the pupils and palpebral apertures). Increase of the blood pressure has been noted by Naumann and by myself in a few cases. The severe vagus pains, of which these patients often complain, may be partly localised in the sympathetic tracts. In one very marked and obstinate climacteric neurosis under my care, the stools became absolutely colourless when the paroxysms of pain were at their height. The symptoms of enteritis membranacea may develop from this cause. The various acroneuroses, in particular acroparæsthesia (*q.v.*) with its atypical and mixed forms, which have been described by Calabrese and Zingerle,[1] undoubtedly tend to occur specially during the climacteric.

Apart from the participation of the sympathetic nervous system in the general neuroses, there are a number of diseases which are so far characterised by the predominance of vasomotor symptoms of certain kinds and by the peculiarity of their localisation, that they may be comprised within a special group. We speak of the *vasomotor neuroses or angioneuroses*. As—in accordance with the theory of Lewaschew that the symptoms produced by irritation or paralysis of the vasomotor centres are most marked at the periphery of the body—the distal parts of the extremities are as a rule specially affected, the name of *acroneuroses* has been given to the condition. The fact that the distal ends of the extremities are pre-eminently open to the influence of the thermal stimuli which specially affect the vasomotor system may contribute to this localisation.

The vasomotor disturbances which are most prominent are combined as a rule with sensory, secretory, and trophic symptoms. We are only justified in classifying these affections among diseases of the sympathetic in so far that many of the symptoms are transmitted through the sympathetic system. This does not imply that the sympathetic itself is the starting-point of the disease.

Philipsohn, Török,[2] and others rightly advise that the term angioneuroses should not be used too freely, and that it should not be applied to the hæmatogenous dermatoses.

Vasomotor Neurosis of the Extremities (Nothnagel). Acroparæsthesia (Schultze)

Bibliography in Cassirer (*loc. cit.*) and Frankl-Hochwart, "Akroparästhesien," Nothnagel's "Handbuch," xi., second edition, 1904.

The disease is usually observed in women, especially at the *climacteric*, but very rarely before the thirtieth year. Men are occasionally affected.

The causes mentioned, other than the climacteric, are: exposure to cold, the constant effect of cold water or waters of different temperatures upon the hands (washerwomen), overstrain of the hands in sewing, knitting, etc. Anæmia, cachexia, and pregnancy may also apparently be causes. Saundby and Shaw think the condition may be of gastric or toxic, and Sommer[3] of traumatic origin. It has occasionally been attributed to influenza.

Schmidt has referred to its relation to tuberculosis, but he was not apparently thinking of typical acroparæsthesia.

[1] *Jahrb. f. P.*, xix.
[2] *Gyogyiszat*, 1906; *C. f. N.*, 1906. See also Jadassohn, *B. k. W.*, 1904, with bibliography.
[3] *B. k. W.*, 1902.

Cassirer has found from a study of our cases that the affection tends to develop from the neuropathic diathesis.

The *symptoms* are in most cases entirely *subjective*. The patient complains of *paræsthesiæ* in the hands, especially the fingers. Formication, numbness, or a feeling that they are asleep—any of these terms may be used—is most severely felt in the *tips of the fingers*, but in lesser degree it may extend to the proximal parts of the extremities. The feet and toes are only involved in rare cases. The sensations may be so intense as to be *painful*, but rarely so to a marked degree—as in a few cases under my care. These paræsthesiæ are continuous, being seldom interrupted. They are almost always most marked at night and in the morning, before and after rising, so that fine handwork cannot be accomplished in the early morning. The patient tries to ease the hands by rubbing, beating, or warming them. The formication is often more acutely felt when the patient tries to grasp an object. In some cases there is a feeling of cold and stiffness in the fingers. The affection is frequently limited to one hand or to certain fingers.

Objectively we find either no change or merely *slight decrease of the sensibility* of the finger-tips. Cassirer found this in a third of his cases. A radicular extension of the sensory disorder has been noted in a few cases (Pick,[1] Dejerine-Egger[2]). The hypæsthesia may be present at times and absent at others. In rare cases there is evident *pallor* of the fingers. All the other functions are normal. A few of my patients have complained of a rush of blood to the head, of cardiac palpitation, etc. I have occasionally observed vasomotor disorders of another kind, *e.g.* urticaria factitia, in the descendants of these patients.

The affection develops insidiously; an acute development, as in one of my cases where it is said to have come on suddenly one summer, after the patient had placed her perspiring hands upon ice, is very uncommon.

The *course* is chronic. The disease usually lasts for many years, although some cases have a rapid course. The *prognosis* as to recovery is not very favourable, although *spontaneous recovery* or great improvement may occur after many years' duration, as some of my observations show. Dejerine indeed considers this to be the rule. The disease entails no danger to life.

In its first commencement, it may easily be mistaken for some other nervous disease. Tabes dorsalis may begin with paræsthesiæ in the upper extremities, but this is accompanied or soon followed by lightning pains, ataxia of the arms, etc. A careful objective examination will prevent confusion with gliosis and other spinal diseases. Raynaud's disease may commence with paræsthesiæ, but local asphyxia, cyanosis, and finally gangrene develop. Ergotism is discovered from the history. A similar syndrome, described by Rosenbach, is characterised by the presence of tubercles on the end phalanges. Pfeiffer regards the condition as gouty, and identifies it with "Heberden's nodes." There are forms of tetany in which such paræsthesiæ are very marked, but they are associated with increase of the mechanical and electrical excitability of the nerves. Shaw has described mixed forms of acroparæsthesiæ and tetany.

[1] *R. n.*, 1903, and *B. k. W.*, 1906.

[2] *R. n.*, 1904; see also Trombert, *Thèse de Paris*, 1905; Baup, abs. *N. C.*, 1905; Bouchaud, *R. n.*, 1904.

Similar symptoms may occur at the commencement of *acromegaly* (Sternberg).

Hysterical persons not infrequently complain of paræsthesiæ in the hands, but this is only an intercurrent, inconstant symptom, and its character can usually be easily recognised by the effect of mental influence upon it.

The *parœsthesia* described by Berger is not identical with this disease. In it a sensation of formication and deadness, of stabbing or burning, comes on paroxysmally in one or both legs (rarely passing to the arms); it radiates downwards from the hip or upwards from the foot to the thigh. It comes on specially at the beginning of a movement or of walking, and is associated with a feeling of weakness. There may also be a kind of girdle-sensation, but no objective symptoms.

Acroparœsthesia is probably due to a *condition of irritation in the vasomotor centres*, by which the arteries are contracted and the nutrition of the sensory nerve-endings in the extremities impaired. There is hardly any support for the view that the symptoms are due to slight neuritis of the terminal nerve branches, especially as pressure-points, tenderness to pressure, and other objective signs are absent. The radicular distribution of the disturbance of sensibility is some support for the view that the disease originates in the spinal cord.

Cassirer would distinguish two types: the simple disease (Schultze's form), which represents a sensory neurosis, and Nothnagel's variety, consisting of vasomotor and sensory disturbances, and representing a vasomotor-sensory neurosis.

Arsenic, *phosphorus*, *strychnine*, and *iron* have been recommended, as well as galvanisation of the medulla oblongata, the cervical cord and sympathetic, and *local faradism*. *Quinine* (3 to 5 grs., given just before bedtime) has sometimes proved beneficial, as I have also found. Sinkler approves of ergotin. Overstrain of the hands, the use of cold water, etc., should be forbidden.

Acute Circumscribed Œdema of the Skin (Quincke). Hydrops Hypostrophos (Schlesinger)

Bibliography: Quincke, *M. f. Dermat.*, 1882; Strübing, *Z. f. kl. M.*, ix.; Joseph, *B. k. W.*, 1890; H. Schlesinger, *W. kl. W.*, 1898; Review in *C. fr. Gr.*, i.; *M. m. W.*, 1899; F. Mendel, *B. k. W.*, 1903; Cassirer, *loc. cit.*: Vervaceck, *Bull. de l'Acad. méd. de Belgique*, 1903; Quincke-Gross, *B. k. W.*, 1904; Ebstein, *V. A.*, Bd., clxxiv.; Diller, *N. Y. Med. Journ.* 1906; Valobra, *Nouv. Icon.*, xviii.; Armand-Sarvonat, *Gaz. des hôp.*, 1905.

The disease generally affects *young* persons, men more frequently than women. It is not uncommon in children. It takes the form of paroxysmal attacks of *circumscribed œdematous swelling* of the skin and subcutaneous tissue, sometimes also of the mucous membrane. Rounded swellings, usually of two to ten cm. in diameter, appear. These stand out prominently from the skin, and are either pale in colour or much redder than the surrounding skin. As a rule there is no itching or pain. The swellings develop simultaneously at different parts of the body and disappear in a short time, but they may recur repeatedly and in rare cases leave permanent traces. They are usually circumscribed, but the whole of one extremity, one side of the face, the scrotum, etc., may be involved.

The *tongue*, *lips*, *larynx*, *pharynx*, *conjunctiva*, and probably the *stomach* and *intestine* may be the seat of the œdema. The lips, eyelids, and cheeks are most commonly affected. *Effusion into the joints* is an occasional accessory symptom. In a case of this kind which I saw, the sudden swelling of the joints simulated an attack of gout. Schlesinger describes intermittent swelling of the tendon sheaths. Quincke and Gross, and M. Herz (*C. f. i. M.*, 1908) have recently observed swelling of the periosteum due to this condition.

The general health is not affected as a rule, but the swelling of the mucous membranes may cause troublesome conditions, *e.g. gastro-intestinal symptoms*, such as nausea and *periodic vomiting*, due to " urticaria interna " (Quincke, Dinkelacker, Strübing, etc.). Diarrhœa has been attributed to this cause. Harrington [1] describes severe " abdominal crises " of this character, laparotomy showing local œdema and hæmorrhage in the small intestine. Œdema of the glottis is very rare, but it has caused death in a few cases (Osler, Griffith, Sträussler [2]). F. Mendel has observed a hereditary familial form of the disease, six persons having died in four generations with the same symptoms of suffocation. In a case of Higier's, the œdema of the soft palate brought on attacks of pseudo-croup. Œdema of the uvula has been described by Quincke and Gross. Hæmorrhages from the mucous membranes (bladder, bronchi, stomach, etc.) rarely occur. Joseph and Valobra have observed a case in which there was paroxysmal *hæmoglobinuria*. Albuminuria may also occur, and in a case under my observation it led to a diagnosis of kidney disease. Rise of temperature is an unusual symptom. Quincke is inclined to ascribe certain forms of serous meningitis to acute circumscribed œdema of the brain. It is uncertain whether acute circumscribed œdema may cause brain symptoms. I have seen an apoplectic attack occur in a chronic variety of this disease, which was possibly of vasomotor origin. Loewenheim [3] has seen a similar case. I suspect that an affection of the optic nerve [4] may develop from this cause, but this can only be assumed with much caution and reserve. Schlesinger supposes that bronchial asthma may be produced in this way, and Wright has reported one such case. Transient œdema of the lungs has been described by Quincke-Gross.

Schlesinger further states that acute relapsing œdema of the eyelids (Fuchs), acute relapsing exophthalmus (Gruss), nervous sniffing, and other paroxysmal nervous troubles belong to this class. It has already been noted that Ménière's disease may be due to similar processes in the labyrinth.

The persons affected are almost always of a *nervous* disposition, and heredity is an important factor in the etiology. A familial occurrence is described by Quincke, Strübing, Osler, Schlesinger and F. Mendel. A combination with *hysteria*, *neurasthenia*, *exophthalmic goitre*, *urticaria*, etc., has often been observed. Rad [5] has seen epileptic attacks occur in the course of the disease, but his case is not above question. The condition is evidently nearly allied to urticaria, and was formerly described as giant-urticaria (Milton). Even yet some authors regard the two conditions as identical. I have treated an acute œdema of the hands and

[1] *Boston Med. and Surg. Journ.*, 1905. [2] *Prag. med. Woch.*, 1903. [3] *B. k. W.*, 1903.

[4] Handwerck (*M. m. W.*, 1907; abs. in *N. C.*, 1908) has published a case of this kind. In a careful review of the literature my statements should not have escaped his notice (compare also p. 715, and the remarks in the earlier edition of this textbook).

[5] *M. m. W.*, 1902.

feet associated with hyperidrosis and inequality of the pupils in a young man whose father had suffered for twenty years from urticaria. Alcoholism may apparently bring on the condition. The uric-acid diathesis and various intoxications (Oppenheimer), especially from the intestine (Le Calvé, F. Mendel, Quincke-Gross, Quincke [1]), have been regarded as the cause. It is not yet known whether the affection may have an infective origin. Some ground is given to this supposition by an endemic outbreak which Loewenheim observed in Lower Silesia.

Exposure to cold, mental emotion, and trauma are regarded as the exciting causes. A few observations point to the importance of these factors. Thus in one case the swelling appeared only on the exposed parts of the body. Cassirer thinks local injuries and thermal influences are of special importance. In one case the condition followed a fright, in another the sting of an insect. The attacks often coincide with menstruation (Modino, Börner, etc.). In one case they were brought on by irritation of the nerves of taste. Staying at the sea-side also seems able to produce the condition.

Various theories have been advanced to explain the occurrence of circumscribed œdema. A local *venous spasm* has been assumed as a cause. Some writers think that the nerves have a direct influence on the capillary cells, and may excite them to the secretion of lymph, a view which seems to be upheld by the experiments of Heidenhain and Starling.

See also Baum (*B. k. W.*, 1905); we would likewise refer to the interesting investigations of Kreibich (*D. m. W.*, 1907), which relate to the production of angio-neurotic œdema by the use of the faradic brush.

Acute circumscribed œdema is an *intractable* affection, as relapses are frequent and the trouble may thus last for many years. Life is very seldom endangered thereby.

As regards treatment, the most important points are attention to the general health, and strengthening the nervous system. Hydrotherapy (cold rubbings, river-baths, etc.) is specially useful. The digestion should be regulated. Aperients may be needed. Atropin and quinine have been given. I have found the latter very beneficial, as it cured three of my cases and produced great improvement in other three. Valobra reports cure by injections of atropin. Kreibich [2] recommends arsenic, in addition to the avoidance of exciting causes, especially thermal influences. Psychotherapy may also be of service.

For the alleviation of the local troubles, the drugs recommended for urticaria, *e.g.* bromocoll, euguform, may be used. (Compare Joseph [3]).

Œdema of a more *constant* nature, with periodic exacerbations, may have a neuropathic origin (chronic, neuropathic œdema). This includes so-called hard, traumatic œdema (Sécrétan-Vullier, Grünbaum [4]) and the allied forms. In a patient of mine, white œdema of the hands, nape of the neck and feet, with from time to time marked exacerbating pain, was a chronic condition; she also suffered from nervous tinnitus aurium. Higier has reported similar cases. H. Meige [5] describes a chronic form of

[1] "Über Hydrops toxicus," *B. k. W.*, 1906.
[2] *D. m. W.*, 1903.
[3] *A. f. Kind.*, Bd. xxxviii.
[4] *D. m. W.*, 1903. See also Étienne, *Nouv. Icon.*, xx.; Cheinisse, *Sem. méd.*, 1903.
[5] *Nouvelle Icon.*, 1893.

this kind as "*trophœdème.*" It is mostly localised in the lower extremities, but may affect the upper limbs or face. Pallor, hardness, and painlessness are its characteristics, and among these Meige and Debove include segmentary distribution.

Several members of one family may be affected (cases of Meige, Lannois, Lortat-Jakob), or a single member (Vigouroux, Hertoghe, Mabille); a congenital form has been described (Nourre). The predisposition is perhaps always congenital. See also Lannois (*R. n.*, 1904), Roué (*Thèse de Lyon*, 1904); Dopter (*Gaz. des hôp.*, 1905), Sicard and Laignel-Lavastine (*Nouv. Icon.*, xvi.), Sainton-Voisin (*Nouv. Icon.*, xvii.), Étienne (*Nouv. Icon.*, xx.), Londe, Valobra, etc. Meige and Dide (*Nouv. Icon.*, xvi.) speak also of a pseudo-œdema of catatonia. Our knowledge of *general idiopathic œdema* is still very defective. Staehelin (*Z. f. kl. M.*, Bd. xlix.) has reported a case of this kind with a fatal termination. Eschweiler (*D. m. W.*, 1905) has described a form of obscure origin.

Intermittent Articular Dropsy [1]

This is a very rare affection, first described by Moore. Its chief symptom is *swelling of a joint* caused by an effusion of fluid. In most cases the *knee-joint* is affected on one, less often on both sides, but the swelling may involve other joints, including the spinal column and the temporo-maxillary joint (Féré). It comes on *periodically*, at regular intervals every second day or week (every eighth or thirteenth day, or in intervals of four weeks, or rarely of some months). The patients can sometimes predict the day on which it will appear. In a few cases it coincides with menstruation (Benda). The skin over the joint is as a rule neither red nor hot; the temperature is normal, and pain is not always present. Slight fever occurs only in a few cases. Tachycardia is more often observed, and vertigo, vomiting, polyuria, etc., are occasional symptoms.

The attack lasts, as a rule, from three to eight days. The effusion then becomes absorbed, and the condition is once more normal. In one of my cases the swelling always lasted exactly twenty-four hours. It may be an independent disease or come on as part of a *general neurosis*, or in combination with *exophthalmic goitre*, *angina pectoris*, etc. Rheumatic influences and injuries have also been regarded as causes.

In one case attacks of flushing of the face, polyuria, and hyperidrosis alternated with the dropsy. In another, in which the dropsy developed in the course of exophthalmic goitre, the symptoms of the latter disappeared when the swelling in the joint came on. In one of my patients a periodic pain in the thigh, occurring at regular intervals, apparently formed an equivalent of the previous dropsy. Cutaneous hæmorrhages appeared in the course of the disease in a case observed by Burchard, and the articular swelling alternated with attacks of asthma. Féré saw swelling of the knee-joint occur in a hysterical morphinomaniac during abstinence; it disappeared in a few minutes after an injection of morphia. In another case the swelling of both knee-joints was associated with œdema of the thigh. He also described a chronic dropsy.

Weiss mentions a variety of this affection, characterised by a certain degree of persistent dropsy, with periodic exacerbation. In two neuropathic individuals I have observed a relapsing *iritis*, associated with hemianæsthesia of the same side, which in its mode of onset reminded me very much of intermittent dropsy.

[1] Literature in Schlesinger, "Hydrops hypostrophos und Hydrops articul. intermittens.," *Mitt. aus. d. Grenzgeb.*, v.

This is undoubtedly a *nervous* affection, which has nothing to do with acute articular rheumatism, although micrococci are said to have been found in one case. Its relation to malaria is also doubtful. Schlesinger, who has carefully studied the condition (and was able to collect fifty-five cases), includes it with acute, circumscribed, cutaneous œdema, and with hydrops hypostrophus.

Crépin (Paris, 1903) has devoted a thesis to neuro-arthritic œdema. Linberger (*Bruns Beiträge*, xxx.) treats of it from the surgical point of view.

The disease is very *persistent*. In one case it occurred at intervals of nine days during a period of eighteen years. *Electricity*, *salicylic acid*, *quinine*, *arsenic* (H. Köster reports definite cure from arsenic in one case), *ergotin*, the Carlsbad method of aperient treatment, etc., have been recommended. Surgical measures (puncture, iodine injections, etc.) should only be used in case of necessity, but Wiesinger (*D. m. W.*, 1903) has been successful with puncture followed by injection of iodoform and glycerine. A few other cases of this kind have been recorded. Some cases call for the use of hypnotism or similar mental treatment.

Symmetrical Gangrene
(Symmetrical Local Asphyxia; Raynaud's Disease)

Literature in Cassirer, "Die vasomotorisch-troph. Neurosen," Berlin, 1901. Among the more recent papers those of H. Strauss, *A. f. P.*, xxxix.; Bonnenfant, *Thèse de Paris*, 1904; Sommelet, *Thèse de Paris*, 1905; Sarvonat, *Gaz. des hôp.*, 1907, etc., should be studied.

Symmetrical gangrene may occur independently or appear in the course of other nervous diseases, such as *hysteria*, *traumatic neuroses* (*q.v.*), *tabes dorsalis*, *syringomyelia*, *disseminated sclerosis* (Strauss), *tumours* of the spinal cord and its roots, *epilepsy*, *exophthalmic goitre*, etc. Courtney states that it is comparatively common in mental diseases. It seems to be entirely or at least chiefly due to the *neuropathic diathesis*. Anæmia and exhaustion, and still more congenital narrowness of the aorta, increase the predisposition. Young people, mostly of the female sex, are most liable to the disease; men and older people are less often affected, but out of sixteen patients under my observation, ten were males. Infants may also be attacked. Cassirer thinks most of the cases occur before the age of fifteen. The most important *exciting cause* is *mental emotion* (fright, etc.). Exposure to cold and suppression of the menses are other causes. Some writers, such as Dekeyser[1] and Broca,[2] are inclined to regard it as being related to chilblains. Trauma may also bring it on. An interesting case of this kind, in which it followed an injury to the head, has been described by Schäffer. It has occasionally been observed after infective diseases (typhoid, influenza, erysipelas, and, according to Seidelmann,[3] after pneumonia), in association with nephritis, pericarditis, etc., and after syphilis. In a few cases it has been thought to be related to tuberculosis (Seé, Burkhart, Rénon, Bonnenfant). In one it was attributed to morphia and chloral poisoning, in another to chronic lead-poisoning. Some writers, *e.g.* M. Weiss, Monro, Cassirer (see below), would only regard as Raynaud's disease those cases in which it has an independent existence, and would exclude from this class those analogous affections

[1] *Journ. méd. de Bruxelles*, 1902. [2] *R. n.*, 1902. [3] *Z. f. N.*, xxvii.

which occur in the course of spinal-cord diseases, infective diseases, etc. Others are inclined to take a wider conception, and even to include diabetic gangrene.

The disease almost always comes on in paroxysms. The *attack* commences with paræsthesiæ, a feeling of formication and deadness in the fingers, possibly also in the toes. The fingers become white and cold, or *waxen-white*, like those of a dead person (*local syncope, regional ischæmia*). The prick of a pin does not draw blood. Violent pain in the whole extremity, but specially in its distal parts, may be present for some days or weeks before the attack, increasing with its onset, and sometimes becoming extremely acute. Hyperæsthesia and hypæsthesia may be present at this stage.

Local syncope may pass off without leaving any trace, or it may be followed within a few minutes, hours, or later, by *regional cyanosis* (local asphyxia). This is occasionally present from the first. The skin at symmetrical parts of the hands and feet, fingers, and toes, almost always in the

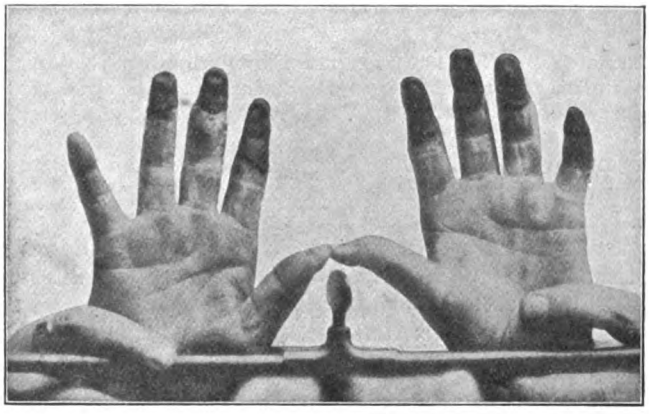

FIG. 423.—Symmetrical gangrene. (After Dehio.)

terminal phalanges, becomes first *blue-red* in colour, then deep blue, blue-black, and finally black. The pain becomes unbearably intense. The epidermis may show vesicles and slight hæmorrhages (Hochenegg, Criegern [1]).

The cyanosis is directly followed by *gangrene*, or the blue-black colour becomes red, and the coldness decreases (regional rubor), or redness may from the first be present instead of cyanosis. Weiss attributes these symptoms to spasm of the vaso-dilators. Even in this stage recovery is possible. The gangrene usually commences with the development of *small black spots* or vesicles filled with bloody serum; these burst and leave a superficial *black crust* which gradually exfoliates, or an ulcer which slowly heals over. But the gangrene may extend into the deep parts, the whole or great part of the phalanx becoming mummified (Fig. 423). It is very rare for a considerable part of the limbs to be involved, as in a case of Barlow's. The dead tissue is bounded by a line of demarcation, and drops off in the course of a few months, while the stump gradually heals up. The suppuration is usually slight.

[1] *D. m. W.*, 1903.

The whole process runs an afebrile course. The intense pain, which prevents sleep, has a bad effect upon the general condition. *Mental disorders* may precede the attack; deep depression very often precedes or accompanies it. Gastric disturbances, *e.g.* loss of appetite, vomiting, abdominal pain, are not uncommon at this stage.

The *sensibility* for all kinds of sensation, or for some of them only (especially for pain and thermal stimuli), is greatly diminished in the affected parts. *Slowness of sensory conduction* has also been observed. The movements are usually limited, stiff, and slow. In a few cases there was *atrophy* of the small muscles of the hand, hypertrophy of the epidermis, and changes in the nails. *Trophic* disorders in the joints, *e.g.* acute synovitis, spreading over many joints and accompanied by swelling of the soft parts in the neighbourhood, are comparatively seldom seen. It has been proved by X-ray examination that the trophic disturbances extend to the bones (Beck, Oppenheim-Cassirer,[1] Lippmann,[2] Wolff,[3] Levy-Dorn, Albers-Schönberg,[4] etc.). The temperature of the skin may during the stage of local asphyxia fall far below that of the surrounding medium.

H. Curschmann (*M. m. W.*, 1907) has proved by plethysmographic examination that in severe cases the normal vascular reflexes are absent, and that pathological, paradoxical reflexes may appear, such as contraction of the vessels on the application of heat, etc.

In addition to the local changes and disturbances of the general condition, symptoms of involvement of the *special sense organs* may occur in rare cases. Thus *visual disorders* have sometimes been present during the paroxysm, and have been attributed to spasm of the retinal arteries. *Deafness*, tinnitus aurium, affections of the sense of taste, etc., may be transient symptoms. *Albuminuria*, *glycosuria*, *hæmaturia*, or *hæmoglobinuria*[5] have been mentioned as symptoms. M. Weiss describes a condition of collapse which he attributes to spasm of the coronary arteries. Signs of *paralysis of the sympathetic*, especially oculo-pupillary symptoms, have occasionally been noted. Aphasia, epilepsy, and paralysis of cerebral origin in the extremities are unusual symptoms, and are also attributed to spastic conditions in the vessels (Osler, Oppenheim).

As a rule the gangrene involves the terminal phalanges of certain fingers and toes in a symmetrical manner. It is rare for all of them to be affected. Asymmetrical extension or unilateral development of the gangrene is an exceptional occurrence. Strauss therefore prefers the term angiospastic gangrene. It is not uncommon for the gangrene to spread to the middle and basal phalanges, and it may extend to the tip of the nose, the ear, nates, thigh, etc., and even, in rare cases, be limited to the tip of the nose and the ears (Declouz, etc.). The formation of ulcers along the frænum of the tongue has also been described.

The attack as a rule lasts for two to four months. The disease is generally limited to a single attack, but it may recur several times, the whole process lasting, with remissions, for several years. Slighter vasomotor disorders are usually present in the intervals. Naunyn[6] mentions

[1] S. Fürnrohr, "Die Röntgenstrahlen im Dienste der Neurologie," Berlin, 1906.
[2] *D. m. W.*, 1900. [3] *B. k. W.*, 1904. [4] *D. m. W.*, 1906.
[5] Cassirer points out the great similarity between paroxysmal hæmoglobinuria and Raynaud's disease.
[6] *D. m. W.*, 1901.

that this chronic form, lasting for many years and developing into sclerodermia, is particularly common in Strassburg, but a combination of Raynaud's disease with sclerodermia has also been observed elsewhere. A transition form between Raynaud's disease and erythromelalgia, which will be discussed presently, may also occur (Rolleston).

Multiple, painful whitlows have occasionally developed in the intervals between the attacks (Mills).

We know nothing for certain as to the *cause* of the disease. It is probably a vascular spasm affecting both the arteries and veins. *Neuritic* changes have also been found in the nerves of the fingers (Pitres and Vaillard), but these are probably secondary to, or due to the same cause as the other symptoms of the necrosis (Dehio, Cassirer), and they are often absent (Dufour). Dehio [1] observed *endarteritis* and *endoplebitis* of the small vessels. Changes, either in the peripheral vessels or nerves, or in both of these, are most frequently, though by no means always present. There is also no doubt that Raynaud's disease may occur in the course of tabes dorsalis and syringomyelia, but in the great majority of cases there is no disease of the central nervous system. The changes in the spinal cord, found in one case by Lyle and Greiwe,[2] were regarded by them as secondary (due to arteritis).

The most probable explanation is that various processes (organic diseases or conditions of infection or intoxication), localised at certain parts of the spinal cord (posterior and *lateral* grey matter ?), may give rise to the syndrome, and that typical cases, in which the condition appears independently, are due to slight, reparable changes. Raymond has expressed the opinion that the condition is a central vasomotor neurosis. Cassirer has adopted this view, but has modified it by asserting that the disease is in most cases an independent one, and is only occasionally a link in the chain of symptoms of diseases of another kind (spinal disease, neurosis). He thinks that the vasomotor tracts and centres are in a condition of exaggerated excitability, due to congenital disposition or to repeated injurious influences (cold, infection, intoxication, etc.). It is doubtful whether the syndrome may have a neuritic origin (Pitres, Vaillard, Hochenegg, Lancereaux, Spiller [3]). Cassirer thinks that we have as yet no proof of such a possibility.

We might also imagine it to be a disease of the sympathetic, the effect of the spinal cord disease being (here as in erythromelalgia) to suppress the inhibitory and regulating influences, and thus to leave the sympathetic ganglia to exercise their functions without any control.

Ehlers [4] has suggested and tried to prove that this and the allied tropho-neuroses are due to ergot poisoning, but without any justification. Zenner has stated the interesting fact that he has seen symptoms of local asphyxia develop after prolonged use of *caffeine* in a daily dose of 1·0 g. (15·5 grains). Phisalix reports having produced the affection experimentally.

In making a *differential diagnosis* we should remember that symptoms of *local syncope* do not justify a diagnosis of symmetrical gangrene, as so-called "dead fingers" not infrequently occur in healthy, neuropathic, or anæmic individuals, or those with kidney disease, etc., unaccompanied

[1] *Z. f. N.*, iv. [2] *Philad. Med. Journ.*, viii. [3] *Journ. Experiment. Med.*, 1900.
[4] "L'ergotisme," *Encyclopéd. scientif. des aide-mémoire.*

by any further symptom. Combinations of local asphyxia with melanodermia, vitiligo, and disturbances of cardiac innervation, as described by Merklen, Gouget, and others, may also be difficult to diagnose. Adler has published an interesting case in which the second to the fifth fingers became waxen-white when cold water was applied to them, and cyanosed and swollen under the influence of hot water. There are also some people the skin of whose hands and feet becomes markedly cyanosed when exposed to cold, and in this condition the movements may, for some time, be impaired, although gangrene never develops.

Raynaud's disease can hardly be confused with so-called "clubbed fingers," which is a chronic, and, in our experience, an independent condition; but this mistake has been made in a few cases (Stöltzing).

According to the observations of Berent (*B. k. W.*, 1903), Grödel (*M. m. W.*, 1906), and Bernhardt (*B. k. W.*, 1906), aneurisms of the subclavian or axillary arteries may by pressure on the plexus give rise to unilateral gangrene.

The condition is well characterised by the youthfulness of the patient, the symmetry of the lesions, the sensory, motor, trophic, and other symptoms, and the absence of any cardiac or vascular disease. There are, however, transition cases between this disease and sclerodermia, acroparæsthesia, and the other angio-neuroses. It may also be very difficult to diagnose from so-called *spontaneous gangrene*, which occurs in middle life and occasionally in youth or childhood, and which Haga,[1] in the observations which he made in Japan, attributed to gummatous arteritis. Very probably race, climate, and nicotine poisoning all help to produce it (Wulff). As regards the relation of this affection to so-called intermittent claudication, see pp. 586 *et seq.* Virchow has already pointed out the rôle played by disease of the vascular system in this form of gangrene, but he has also recognised that spastic ischæmia is a factor which aids in the production of the necrosis. Among the recent works on this subject we would mention that of Matanowitsch.[2]

Diabetes and *nephritis* do not cause Raynaud's disease, but it may be combined with nephritis, as both affections may have a common cause and basis (Gibert). The development of local asphyxia and nephritis or albuminuria from the same (vasomotor) origin has been assumed by Barré [3] and others. It is by no means always easy to determine whether symmetrical gangrene is a primary disease or one of the symptoms of an affection of the spinal cord, in particular of gliosis.

It is difficult also to classify the "multiple neurotic gangrene of the skin," which occurs in nervous, usually hysterical persons, and consists in the formation of multiple necrotic lesions. The development of these lesions is preceded by local paræsthesiæ (burning, pricking, etc.), and sometimes by pain. The necrosis is mostly superficial, but keloids may develop in the cicatrices. Vesicles and bullæ sometimes accompany the necrotic process, and the prick of a pin is specially apt to lead to the formation of bullæ with serous contents. There are apparently no changes in the vascular system. The disease has been described as gangrenous urticaria (Rénaut) and as gangrenous herpes (Kaposi). Many authors regard it as a symptom of hysteria, due to self-mutilation. Rona, for instance (*Orvos. Hetil.*, 1905), assumes that it is always an artefact. Cassirer, who summarised the published cases, is, however, not inclined to accept this view, although there is no doubt that similar cutaneous lesions, of artificial production, have been observed in hysterical persons; he regards the condition rather as related to Raynaud's

[1] *V. A.*, Bd. clii. [2] *Beitr. z. kl. Chir.*, xxix.
[3] *Thèse de Paris*, 1903. See also Calonne, *Thèse de Paris*, 1904.

disease, and this is also indicated by an observation of Adrian's. Kaposi points out that the shining of the deeper necrosed portions of the skin through the intact epidermis is evidence in favour of the spontaneous occurrence of the gangrene. Zieler (*Z. f. N.*, xxviii.) also lays stress upon the fact that non-artificial gangrene commences in the deeper layers of the cutis. Doubtful cases can only be decided by histological examination.

Dinkler (*A. f. Dermat.*, 1904) also believes in the neurogenic origin of the disease.

Cassirer has further endeavoured, on the basis of cases observed by himself, Oppenheim, Nothnagel, Souza-Leite, etc., to differentiate from Raynaud's disease a condition which he names "*acrocyanosis chronica anæsthetica*," and in which the chief symptom is *gradually* developing asphyxia of the ends of the extremities, associated either with sensory or trophic disorders. It differs from Raynaud's disease mainly in the absence of paroxysms. This form has also lately been discussed by Péhu,[1] as "acrocyanose chronique hypertrophiante"; his view that it is a tuberculosis of the skin is not borne out by our observations.

Claude (*R. n.*, 1906) has seen the condition arise from, or in association with hysteria. See also Mosse (*B. k. W.*, 1906), Brissaud-Halbron (*Arch. gén. de Méd.*, 1903).

We are unable as yet to determine whether the chronic cyanosis, with marked increase of the red blood corpuscles and of the hæmoglobin content and enlargement of the spleen, described by Rendu-Widal, Vaquet, and Osler (*Amer. Journ.*, 1903), belongs to the conditions included in this group. Among the further contributions to this interesting affection, also known as polycythæmia, see those of Türk, Weintraud (*Z. f. k. M.*, Bd. lv), Kraus (*B. k. W.*, 1905), Bence (*D. m. W.*, 1906), Senator (*D. m. W.*, 1906), Hirschfeld (*Ther. d. Geg.*, 1906), Preiss (*Mitt. a. d. Grenzg.*, xiii.), etc. The notable observations of Koester (*M. m. W.*, 1906), and Cassirer and Bamberger (*D. m. W.*, 1904), whose case came from my practice, tend to confirm the suspicion which I expressed in the previous editions, that the disease is related to the nervous system. Saundby (*Brit. Med. Journ.*, 1907) is of the same opinion.

Mention has occasionally been made of an enterogenous cyanosis, *i.e.* one due to intestinal auto-intoxication (Stokvis, Palma, Hijmans, Cheinisse, *Semaine méd.*, 1905).

Persistent or frequently repeated vasomotor disturbances may lead to atrophy of the muscles supplied by the affected vessels. We have found this in several instances, and Luzzatto [2] has described some such cases.

As regards the relation between Raynaud's disease and sclerodermia, consult the chapter on the latter.

In lepra mutilans the gangrene simultaneously involves several phalanges, which drop off painlessly. The specific changes of leprosy confirm the diagnosis.

The *prognosis* is on the whole favourable. The disease does not itself cause death. Relapses are specially to be expected when the symptoms are due to an incurable disease, but even in other cases the attacks may often recur. Septic infection may in rare cases be the cause of death.

The object of *treatment* should be to improve the general health, to protect the patient from excitement, and to strengthen the nervous system in general. Therefore a stay in the mountains or at the seaside, or hydrotherapeutic treatment may, by building up the nervous system, remove the cause of the disease. In one of our cases, the symptoms were promptly cured by the patient spending the cold season at Meran. During the attack, mental and physical rest are urgently required. If the pain is very severe, narcotics (chloral or morphia) should be given. Subcutaneous injections, especially at the parts affected by the vasomotor and trophic disorder, are somewhat risky, as they may

[1] *Nouv. Icon.*, xvi.

[2] *Z. f. N.*, xxiii.

produce local gangrene. The affected parts should therefore not be excited, as even the faradic brush may increase the vascular spasm. Irritating ointments should be absolutely avoided.

Gentle massage and *dry warmth* have a soothing effect. *Lukewarm hand* and *foot baths*, *Priessnitz's bandages*, and *galvanic treatment* of the spinal cord and sympathetic have been recommended. Amylnitrite has not been found to be useful. Nerve-stretching has recently been tried (see *e.g.* Bovis [1]).

When suggestion has proved to be of service, as in the cases described by Tesdorpf, Souques, and Gilles de la Tourette (see Le Gall's [2] thesis), the condition has probably not been one of typical Raynaud's disease, but a manifestation of hysteria, or an artificial production.

Erythromelalgia

Literature in Cassirer, *loc. cit.*

The condition to which Weir Mitchell has given the name of erythromelalgia, and which Lannois has carefully described, is characterised chiefly by *pain* and *redness of the skin at the distal parts of the feet*, less often of the hands or of all four extremities. As a rule pain is the first symptom. It may come on suddenly, or be preceded by general discomfort or fever. It sometimes follows over-exertion. It occurs in the feet and toes, especially in the balls of the great toes and on the heels, and in the hands and fingers, but it may also pass into the proximal parts of the limbs. It is rapidly followed by redness, most marked in the end phalanges of the toes and fingers. The pulp especially is reddened and *swollen*. The patient complains of a painful feeling of heat, and the temperature of the skin is objectively raised. The blood-vessels pulsate distinctly. There is sometimes local hyperidrosis, and occasionally *small nodules* develop on the parts most affected. Sensibility is not as a rule markedly impaired, but there may be hyperæsthesia or slight hypæsthesia.

The redness, swelling, and pain vary in intensity. At first, the pain is transient, occurring specially in the evening and when the foot is tired. Later, it may be stationary or merely remittent. It is seldom mild, but is generally severe, and may increase to "real torture." It is soothed by the recumbent position and cold, and is aggravated by standing, walking, hanging the foot, and by heat. The swelling in particular is increased by walking. In standing, the feet become redder and redder, the vessels pulsate strongly, and the skin of the extremities may assume a dark purple or cyanotic colour. The veins also stand out prominently. Hyperidrosis is a common accessory symptom. If the symptoms are very severe, the patient cannot walk, and carefully avoids touching the ground.

The health may be otherwise normal, but *accessory* symptoms are generally present. In most cases there is *headache*, *vertigo*, *palpitation*, *attacks of faintness*, and general weakness. Tachycardia frequently occurs. Dilatation of the vessels and cyanosis may appear at other parts. In one case there was paroxysmal swelling of the testicles and priapism. Gangrene has developed only in a few cases, as Sachs and Wiener have reported, but it is doubtful whether such cases ought to be regarded as *erythromelalgia in Weir Mitchell's sense*. Other trophic disturbances, *e.g.* atrophy or thickening of the skin, swelling of the bones, changes in the nails, etc., may occur. In a few cases increase of the erythrocytes in the

[1] *Semaine méd.*, 1904.

[2] Paris, 1902.

blood has been noted (Schmidt); compare also Osler's observation in the previous chapter). Numerous other cases show that erythromelalgia may occur in the course of a central disease of the nervous system, such as *hemiplegia*. An *atrophic* condition in the muscles of the extremities was present in a few cases. In one of our patients an X-ray examination revealed diffuse atrophy of the bones (see Fürnrohr, *loc. cit.*). A combination with muscular dystrophy, or with symptoms of cerebral tumour, has also been observed (Eulenburg). Auerbach has recognised the syndrome in a case of disease of the cauda equina. The relation to diseases of the spinal cord (in particular to disseminated sclerosis) has been specially pointed out by Collier.[1] Landgraf describes a combination with myxœdema.

The disease is a rare one. Among twelve thousand patients in my polyclinic, only two had erythromelalgia (according to statistics up to the last edition of this book). In my private practice I have only seen six cases in the course of fifteen years. Men are mostly affected. *Exposure to cold* is the chief cause, and the condition is often said to follow freezing. The *neuropathic tendency* seems to increase the predisposition. In a few cases syphilis has been regarded as the cause. The affection is very *intractable*, extending with remissions and exacerbations over many years, and refusing to yield to treatment. There is possibly also a benign form (Oppenheim, Koppius). The symptoms are more severe in summer than in winter. Nothing is positively known as to the termination of the condition, nor can we say anything definite as to its cause. Some writers regard it as an *angioneurosis*, which to a certain extent represents the antipodes to acroparæsthesia and symmetrical gangrene—an affection of the vasomotor nervous system (the vasomotor centres or sympathetic) which is not produced by marked pathological changes. Cavazzani-Aracci think it a neurosis, with primary involvement of the vasodilators. On the other hand the opinion has been expressed (Eulenburg) that it is due to a spinal disease involving the posterior and *lateral grey matter* of the cord—the intermedio-lateral tract—or that various morbid processes involving this area of the cord may produce the disease. A spinal origin has also been assumed by Lannois-Porot [2] in one of his cases. The condition has also been classed with the acroneuroses or acrotrophoneuroses already discussed (Hutchinson, Lanceraux). The possibility that it may be caused by changes in the peripheral nerves has also been pointed out. Weir Mitchell has found marked degeneration of the fine nerve branches in one case. He is of opinion that an alteration of the sensory nerves at any point between the spinal cord and the periphery may give rise to the syndrome. Sachs and Weiner ascribe it to a disease of the peripheral *arteries*, in which they have found marked changes amounting to obliteration. Shaw [3] and Hamilton [4] have also noted changes in the vascular system. Lewin and Benda think that the disease is not an independent one, but is merely a symptom of some organic or functional disease of the central nervous system and of the peripheral nerves (neuralgia, neuritis). Cassirer distinguishes two forms or types: 1. independent erythromelalgia, for which he assumes a central nervous origin—a central, spinal, or bulbar neurosis, with vasomotor, trophic, sensory, and secretory symptoms; 2. a form in

[1] *Lancet*, 1898.
[2] *Rev. de Méd.*, 1903.
[3] *Path. Soc. of London*, 1903; *Brit. Med. Journ.*, 1903.
[4] *Journ. Nerv. and Ment. Dis.*, 1904.

which the symptoms are limited to the peripheral nerves. The cause of this he assumes to be a condition of irritation in the peripheral nerves, involving specially the vasomotor (in particular vasodilator) and secretory fibres.

Treatment.—Faradism and cold have been recommended, and antipyrin, antifebrin, etc., may be given to soothe the pain, Hypnosis was successful in one case of hysterical erythromelalgia (Lévi). We have tried psychotherapy in vain in a patient who also suffered from major hysteria.

Dehio has seen some of the symptoms of erythromelalgia disappear after removal of the ulnar nerve. Weir Mitchell also resected and stretched the nerve with great effect in one case ; in another this operation was followed by death from gangrene.

The severity of the pain has in a few cases necessitated amputation (Shaw).

Other conditions are allied to erythromelalgia, but yet cannot be regarded as identical with it. Pick,[1] under the name of *erythromelia*, described a condition of reddening of the skin, partly circumscribed and partly diffuse, which followed marked dilatation of the veins, and developed on the extensor side of the extremity. It was not associated with pain. Klingmüller differs from Pick in his explanation of this affection. Grawitz [2] has described an allied condition which is specially characterised by the acute development of diffuse atrophy of the skin of the whole extremity. See also Ehrmann.[3]

Johannssen has observed in a child a form of vasomotor neurosis which in many respects resembles erythromelalgia and Raynaud's disease. It has already been stated that erythromelalgia may be combined with or develop into Raynaud's disease (Lannois, P. Weber,[4] etc.).

A patient of Potain's showed the characteristic symptoms of symmetrical gangrene on the limbs of one side of the body, and those of erythromelalgia on those of the other. I have treated a mental case in which there was bright red colouring of the skin of the left hand, which remained persistently as the only symptom.

I have also seen individuals who complained only of redness and cyanosis of the hands, which was always present but increased at times, especially during cold weather, to an unbearable degree (see preceding chapter). In a few cases this was associated, especially during the periods of remission, with hyperidrosis. Some sensory disturbance was present in a few instances.

Cases which correspond to none of these types, but represent mixed and transition forms or varieties, have been described by Calabrese, Zingerle,[5] Schmidt, Brissaud-Londe, Rosenfeld,[6] and Spieler,[7] and have come under my own observation. Fürstner [8] has reported a form of vasomotor neurosis very much resembling erysipelas bullosum. It seems to me that, as Cassirer has said, the difficulties of classification are greater in this region of the vasomotor and trophoneuroses, of which there are so many transition and mixed forms, than in any other.

[1] *M. f. Dermat.*, 1894.
[2] *D. m. W.*, 1903.
[3] *W. kl. R.*, 1905.
[4] *Brit. Journ. of Derm.*, 1904.
[5] *Jahrb. f. P.*, xix.
[6] *C. f. N.*, 1906.
[7] *W. kl. W.*, 1905.
[8] *Mitt. aus d. Grenzg.*, xi.

SCLERODERMIA

Literature in Cassirer, *loc. cit.*

This disease, which occurs at any age, but is specially frequent in middle-aged women (who, according to Kaposi, form three-fourths of the cases), is chiefly characterised by an abnormal condition of the skin and subcutaneous tissue. The latter becomes indurated, shrivelled, and atrophied, so that the skin becomes more and more tense, feels brawny and thick, and cannot be separated from the underlying tissue. Disappearance of pigment and abnormal pigmentation are common accessory symptoms, and there may also be pallor, coldness, cyanosis, and marbling of the skin, or, less frequently, formation of scales.

The process may be circumscribed in the form of spots, or diffuse. The spots, at first isolated or disseminated, as a rule become confluent at a later stage, the disease thus extending over a great part of the skin.

The face, neck, and upper extremities are chiefly affected. The upper part of the trunk is often involved, but the legs seldom are. The disease is usually bilateral, although a unilateral form has been described (Hutchinson, Kaposi). In rare instances it follows the course of certain peripheral nerves, or remains limited to the region innervated by a special spinal segment. Brissaud has laid emphasis upon this fact, and Bruns and Huet-Sicard [1] have observed a case of the kind.

In several cases which I have seen, some part of the body exposed to pressure has formed the starting-point of the disease, from which it has spread to other areas. Thus in two women the region first involved had for a long time been compressed by garters which they wore. A band-like distribution in the frontal region has often been observed (Haushalter, Spillmann, Fournier-Loeper, etc.). In such cases the condition may be very difficult to distinguish from facial hemiatrophy. A classification of the cases according to the various modes of distribution, as Raymond and others desire, is hardly practicable.

Kaposi distinguishes two or three different stages, the *stadium elevatum*, the *stadium indurativum*, and the *stadium atrophicum*. The first of these, in which the skin is œdematous and hypertrophic, generally escapes notice. In the indurative stage the skin is hard, firm, and tense, and has often a glossy, polished look.

The condition, in its advanced stage, cannot be mistaken. The smooth, thin, tense, cold skin, with its unusually prominent veins, abnormal pigmentation, or absence of pigment, its unyielding subcutaneous tissue, etc., is so characteristic that the affection is recognised at the first glance. The atrophy may extend to the deep parts, muscles, and bones, as has been shown by X-ray examination (Dercum, Oppenheim-Cassirer). Swellings and knot-like thickenings also appear in the bones, and the joints may be acutely or chronically affected. Myositic changes may, in rare cases, develop in parts where the skin is not affected. Fasciæ, ligaments, and joints are all involved in the process.

If the hands are involved, the fingers may become stiff and crippled (*sclerodactyly*), the bones thinned—a kind of acromicria—and the muscles indurated, shrivelled, and atrophied. Annular constrictions and mutilations (spontaneous amputation) of the fingers are more rare. Düring

[1] *R. n.*, 1903.

describes these conditions as sclerodactylia annularis ainhumoides. Sclerodactyly may be the only change, or may be associated with other sclerodermic processes. Ulceration and gangrene are not uncommon in the later stages.

A very characteristic look is given to the face by the smoothness and stiffness of the skin, and the shortening of the lips and alæ nasi. The movements of the facial muscles become more and more restricted, and the expression is very rigid.

In a very unusual case under my observation, swelling was the chief symptom. It was of general distribution, and the expression of the face was so changed and grotesque on account of the numerous wrinkles, that at first I took the girl of seventeen to be the mother of the mother who accompanied her.

The subjective symptoms are pain, paræsthesiæ, itching, a feeling of tenseness, and the disorders arising from the restriction of movement. Vasomotor symptoms (local cyanosis, œdema, etc.) may, for a long time, precede the sclerodermic process, and they play an important part in its later course. They often take the form of active hyperæmia. Telangiectasis has been observed in a few cases. Cutaneous hæmorrhages may occur in the course of the disease.

Sensibility is hardly ever affected. Curschmann[1] mentions absence of the normal vasomotor reflex. The resistance of the skin to electrical stimulus is increased in some cases. Muscular atrophy may be combined with sclerodermia, and may even occur at distant parts (C. Westphal). Symptoms of paralysis are rare. We have seen ptosis in one case, and Logetschnikow[2] mentions stiffness of the ocular muscles. Articular affections occur now and again. Spillmann describes epileptiform attacks. Mental depression is a common symptom, and general marasmus develops in severe cases.

With regard to the pathological histology, compare Touchard, *Thèse de Paris*, 1906.

In determining the differential diagnosis, we have found that *dermatomyositis* (*q.v.*) comes most into consideration. Future experience alone will show whether *sclerœdema*—a diffuse œdema in the deeper layers of the cutis and muscular tissue—can be distinguished from sclerodermia, as Buschke[3] thinks, mainly on account of its rapid development and its tendency to recovery. The process of *local panatrophy*, described by Gowers,[4] is very probably identical with the disseminated form of sclerodermia and hemiatrophy.

Sclerodermia may be combined with progressive facial hemiatrophy (Hallopeau, Grasset, Eulenburg, Pelizaeus), the two affections being closely allied. It may also be associated with Raynaud's disease, erythromelalgia, urticaria, exophthalmic goitre, and Addison's disease. Dupré and Guillain[5] have seen the combination with exophthalmic goitre and tetany, and Follet-Sacquépée[6] have in one case observed the combination of Raynaud's disease, tetany (?), and sclerodermia. Krieger[7] has seen sclerodermia follow exophthalmic goitre, and I have treated a young lady for sclerodermia whose sister had exophthalmic goitre. The relation to Raynaud's disease is often so close that a differential diagnosis

[1] *Therap. d. Geg.*, 1907.
[2] *D. m. W.*, 1903.
[3] *B. k. W.*, 1902.
[4] *R. of N.*, i.
[5] *Bull. de la Soc. méd. des hôp.*, 1900.
[6] Abs. *C. f. Gr.*, 1903.
[7] *M. m. W.*, 1903.

cannot be made. Naunyn,[1] Garrigues, and others have published cases of this kind. Hemiatrophy of the tongue in sclerodermia has been described by Chauffard.[2]

Almost all the recent writers on the subject (Schwimmer, Kaposi, Eulenburg, etc.) regard sclerodermia as a trophoneurosis. Raymond gives good grounds for this view, which is also our own. Cassirer thinks that the condition is an angiotrophoneurosis, not due to marked changes in the nervous system. Brissaud ascribes it to the sympathetic. Another theory is that it is caused by a local infection, but this is not confirmed by the results of bacteriological examination (Wolters, Nicolaier, Dinkler, Uhlenhuth). Huismans[3] thinks that a combination of an infection and a neurosis of the sympathetic is necessary to produce the condition. It has also been attributed to the thyroid on the ground of the investigations of Jeanselme, Uhlenhuth, Hectoen, Sachs, and others. Nothnagel is also inclined to regard some poisoning or the falling off of some internal secretion as the cause. Strümpell[4] thinks the hypophysis is the origin of the disease, and Roux[5] (and Lafond), on the ground of the results of one pathological investigation, maintain the relation of one form of sclerodermia to the hypophysis. But we must admit that our knowledge regarding the nature and cause of this disease is quite uncertain.

Pathological examination of the nervous system (Westphal, Chiari, etc.) has had no definite result. Westphal himself did not regard the induration of the brain which he found as the primary disease.

We know little concerning the *etiology*. Prolonged emotion, chill, and hereditary influences have been thought to be causes. Cassirer thinks that the most important factor is the nervous disposition and the effect of intensive rheumatic toxines. An observation of Fürstner's seems to show that a familial form of sclerodermia or some allied vasomotor-trophic affection may occur.

The *prognosis* is grave. Remissions, improvements, and even recovery may possibly occur, but they can hardly be expected in advanced stages. The affection usually progresses gradually, and lasts for many years, the patient dying from marasmus or some complication (which is generally a nervous disease).

Treatment consists in massage, local and central galvanisation, local inunctions (naphthol-salicylate ointment, ichthyol ointment), etc. Salol given internally may be good. Ebstein[6] found baths of acetate of aluminium, massage with powdered boracic or salicylic acids, and the internal use of potassium salicylate to be beneficial. The salts of iodine, thyroidin, and other thyroid preparations have been recommended (Singer, Lancereaux, Sachs, etc.); the latter is uncertain and doubtful in its action (Osler, Notthafft). Menetrier-Bloch[7] and Sachs[8] have found thyroidin helpful, and Schwerdt[9] reports improvement after the use of a preparation made from the substance of the mesenteric gland. Peat and sulphur baths have had a good effect in a few cases, and ichthyol, in the form of baths and internal preparations (calcium sulpho-ichthyolate), has been found efficacious. Mosler has been successful with their use and with cod-liver oil in increasing doses. Brissaud recommends static electricity, and Blocq electrolysis. Hebra saw improvement after subcutaneous

[1] *D. m. W.*, 1901.
[2] *Gas des hôp.*, 1895.
[3] *M. m. W.*, 1905.
[4] *Z. f. N.*, xi.
[5] *R. n.*, 1902.
[6] *D. m. W.*, 1903.
[7] Abs. *R. n.*, 1906.
[8] *Phila. Med. Journ.*, 1902.
[9] *M. m. W.*, 1905.

injection of thiosinamin (one-third to one-half a Pravatz's syringe of a fifteen per cent. alcoholic solution every other day). Scholz, Renzi, and Lewandowsky also report recovery after this treatment.

The local treatment by hot air with Tallermann's or some similar apparatus has also been recommended (Neumann, etc.).

Progressive Facial Hemiatrophy

This disease was first described by Romberg. It is very rare, and almost always affects young people from ten to twenty years of age. It may develop in early childhood, but is very uncommon after the age of thirty. A cause can only be ascertained in a few cases ; injury to the face or skull often precedes the atrophy. In one case the exciting cause

FIG. 424. — Left facial hemiatrophy. (Oppenheim.)

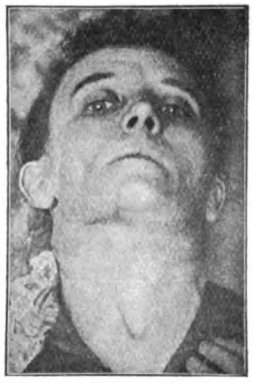

FIG. 425.—Right facial hemiatrophy. Commencement of the process in the submental or upper cervical region. (Oppenheim.)

FIG. 426.—Right facial hemiatrophy in an advanced stage. (After Bruns.)

was apparently the extraction of a tooth (Ziegenweidt), in another the removal of adenoids (Rutten). It has occasionally followed some infective disease (diphtheria, sore throat, erysipelas, measles, typhoid, etc.). Facial neuralgia has been present in several cases, but neuralgic pain is not a common symptom of the condition. In two of my cases there was a marked neuropathic heredity ; in a third, the parents were related to each other, and a sister suffered from congenital dislocation of the hip-joint.

The hemiatrophy develops gradually, and seldom affects the whole side of the face at once. It usually commences at one or more definite points, especially in the orbital region, the lower jaw, or the cheeks (Fig. 424). In one of our cases it was in its initial stage localised in an unusual site, viz., the lower part of the chin or the upper cervical triangle (Fig. 425). O. Fischer [1] has reported a similar localisation.

The affection consists in disappearance of all the tissues. The skin first becomes thin and colourless on account of the loss of pigment, or it assumes an abnormal (brown, yellow, or bluish) colour. Local infiltra-

[1] *M. f. P.*, xiv.

tion may precede the atrophy. The subcutaneous fatty tissue disappears, the skin therefore falls into furrows or grooves, and finally lies close to the bones, which also become atrophied. The process may develop to this extent at one point (Figs. 424, 425) before it spreads further. The atrophy thus affects the skin, subcutaneous tissue, and bones, and sometimes also the muscles of the jaws, face, and even of the tongue. This muscular atrophy is not degenerative, *i.e.* it is not associated with qualitative changes of electrical excitability, nor does it cause any marked impairment of function.

The result of the process is diminution in the size of every part of one side of the face. The bones in particular appear smaller, and the asymmetry is very evident on comparison with the jaw, malar bone, nasal cartilage, etc., of the other side. The eye is deeply sunken, and the cheek furrowed and grooved. The abnormal pigmentation is also characteristic.

FIG. 427.—Right progressive facial hemiatrophy. (After Hirt.)

At an advanced stage the difference between the two sides of the face may be striking (Figs. 426, 427).

The larynx has occasionally been involved (Schlesinger, Körner[1]). In Körner's case the atrophy commenced in the auricle. Höflmeyer found in one case a groove-like hollow in the hard palate and inner side of the upper jaw, which he attributed to atrophy of the pterygoid muscle.

The eyelashes, etc., are usually involved; they fall out or become white. The hair of the head is hardly ever affected, probably because the hemiatrophy seldom extends to the scalp.

The secretion of the sebaceous glands is diminished, but perspiration may be increased. The temperature of the skin is normal. It has been occasionally noted that the skin on the atrophied side does not flush along with the other parts. Herpes was present in one case (Höflmeyer). *Anæsthesia* is uncommon, but I have seen it in two cases on the affected side of the face, but not sharply limited to the distribution of the facial nerve. I have never found tenderness of the fifth nerve branch, but in

[1] *Z. f. Ohr.*, Bd. xli.

some of our cases the region of the highest sympathetic ganglion was painful on pressure.

Salomon [1] describes a case where the hemiatrophy was associated with paralysis of the ocular muscles. Gowers [2] thinks that the process may cause secondary involvement of the facial and other cranial nerves, from narrowing of the bony canals, *e.g.* the aqueduct of Fallopius; but this does not seem to me very clear.

The disease is a progressive one, but sooner or later it comes to a standstill, and it does not imply any danger to life.

The term hemiatrophy is not exactly suitable, as the atrophy sometimes extends to the other side (J. Wolff, Oppenheim). Nor is facial hemiatrophy a name which expresses the whole condition, as the atrophy may involve the shoulder-girdle and indeed one whole side of the body (Raymond-Sicard [3]). This, however, is very unusual, and the type of *hemiatrophia cruciata*, described by Lunz,[4] is still more so. Volhard [5] has published an interesting case of this kind.

Complications with other diseases of the nervous system are not uncommon. It has repeatedly been found along with neuralgia, epilepsy, and psychoses, several times with chorea and spasms of the muscles of the face, jaw, and tongue, and in single cases with tabes, syringomyelia, and disseminated sclerosis. A young woman with advanced facial hemiatrophy consulted me recently, not on account of it, but for a condition of anxiety, imperative ideas, and psychasthenic spasms, for which she desired treatment. I have also seen it combined with congenital paralysis of the ocular muscles. The pupil on the affected side was occasionally dilated or contracted; in one of my cases it was very irregularly contracted, forming an oblique oval. In another case the pupil was not only dilated but almost entirely rigid to light. The combination with symptoms of paralysis and irritation in the sympathetic and other abnormalities (tachycardia, etc.), has been described by Jendrássik [6] and Bouveyron.[7] Bilateral hemiatrophy with sympathetic symptoms is reported by Schlesinger.[8]

In one case, a photograph of which was given in the second edition of this book, there was convulsive tic and typical hemicrania, and the patient also showed on her nose the cicatrix of a cyst, which had emptied itself of its contents. The combination of hemiatrophy with sclerodermia and alopecia has also been described, and Steven has reported an interesting case. Facial hemiatrophy is undoubtedly closely allied to sclerodermia, and the distinction may be very difficult if it is combined with lesions corresponding to the latter at other parts of the body (Brunner, etc.). Thus, I have seen a case in which circumscribed atrophy of the soft parts and bones had developed in the characteristic form upon the back, above the spinal column. The hemiatrophy has also been seen to extend over one whole side of the body. In the case mentioned above, the facial hemiatrophy was associated with lesions of the same kind on the trunk, and also with simple muscular atrophy—without weakness or change of electrical excitability—on the whole of the right leg.

Most writers include this affection with the trophoneuroses and attribute it to the trigeminus (Virchow), the Gasserian ganglion (Bärwinkel), or the

[1] *N. C.*, 1907.
[2] *R. of N.*, 1906.
[3] *R. n.*, 1902.
[4] *D. m. W.*, 1897.
[5] *M. m. W.*, 1903.
[6] *A. f. kl. M.*, Bd. lix.
[7] *Lyon méd.*, 1902.
[8] *A. f. Kind.*, Bd. xlii.

sympathetic nervous system. The results of post-mortem examination are not convincing, and Möbius has rightly disputed the conclusions which have been drawn from them (Mendel [1]). Two of the post-mortem investigations made by Touche [2] in senile cases are of no practical help, but Loebl and Wiesel [3] have again recently found in one case disease of the Gasserian ganglion and the peripheral trigeminus—a proliferating interstitial neuritis. Bitot assumes it to be a primary cutaneous disease. The theory suggested by Möbius [4] that it is due to some local infection or an infective agent conveyed from the tonsils is worthy of note. This view is supported by the fact that the disease often follows local injuries and infective processes in the neighbourhood (angina, abscess of a tooth, etc.). It is, however, also necessary to assume that the unknown infective agent acts chiefly or exclusively upon neuropathic subjects. Brissaud suggests that it is an affection of the peri-ependymary grey matter in the pons and oblongata. Calmette-Pagés [5] agree with him, but there is no proof whatever of their theory. I would not set aside the *sympathetic theory* by any means so decidedly as Möbius does, chiefly because of the fact that the symptoms cannot be brought into accord with an affection of any other part of the nervous system, whilst the sympathetic exercises a great influence upon the trophic condition through its control of the vascular system. Similar forms of facial atrophy have also been sometimes observed in lesions of the sympathetic (*q.v.*). A case of Jaquet's is very interesting in this respect, as the facial hemiatrophy was associated with adhesion of the inferior cervical sympathetic ganglion and the callous, thickened pleura. Clinical observations of this kind, relating to tubercular disease or tubercular pachypleuritis of the apex of the lungs, have been published by Bouveyron,[6] Barrel,[7] Souques,[8] and others. A case observed by Minor of facial hemiatrophy developing after removal of the cervical glands is in favour of this view. The occasional combination of this disease with oculo-pupillary symptoms is in harmony with its being a sympathetic affection, which in pure cases would be localised in the neck or its higher site of origin.

Having observed that the atrophy is often localised at the sites of embryonal clefts of the neck and face, Fischer concludes that there is a *congenital* disposition.

Treatment has no effect, although the galvanic current is recommended. These are the words with which I expressed my view as to the nature of the disease and its treatment in the second edition of this text-book. In the meantime I have made an observation which serves both to prove the sympathetic origin of the disease and to show that, under certain circumstances, it may possibly be improved by treatment. In one quite typical case of this kind (Fig. 324) I found, in addition to marked tenderness to pressure of the corresponding sympathetic, a swollen gland in the region of the superior cervical ganglion. Borchardt, who operated on my advice, found that it was not adherent with the sympathetic, but notwithstanding this, marked improvement took place within five to six weeks after the operation, the sunken appearance of the cheek becoming much less noticeable. It is somewhat difficult to judge of this, as the

[1] *N. C.*, 1888.
[2] *R. n.*, 1902.
[3] *Z. f. N.*, xxxvii.
[4] *Nothnagels Handbuch*, xi. 2.
[5] *Nouv. Icon.*, xvi.
[6] *Lyon méd.*, 1899, and *R. n.*, 1902.
[7] *Thèse de Lyon*, 1901-1902.
[8] *Soc. méd. des hôp. de Paris*, 1902.

patient gained in general strength, and the improvement has, unfortunately, not continued beyond a certain point.

Chipault has removed the sympathetic, but without any result.

Gersuny has attempted to remedy the asymmetry by subcutaneous injections of paraffin, and Luxemburger, Eckstein,[1] K. Mendel, and others have reported similar attempts. It is doubtful whether the result is permanent. On the other hand the operation is not without risk, as an embolism of the central retinal artery has repeatedly occurred (see *e.g.* Uhthoff[2]). Stegmann[3] has obtained excellent cosmetic results with Gersuny's injections of oil and vaselin. A. E. Stein[4] has given a comprehensive account of the matter.

Progressive facial hemihypertrophy (Montgomery), and unilateral and even crossed hypertrophy of the face and the opposite side of the body, may occur in rare cases. Sabrazès and Cabannes[5] have collected from the literature seventeen cases of congenital, and five of acquired unilateral facial hypertrophy, and have added one of their own. Hoffmann[6] and Minor have also described a case. Greig's statistics include thirty-five cases of hypertrophy of the skull and face, and forty-two in which the extremities of one side of the body were excessively developed.

Papers by Thomas, Mouchet,[7] Cagiati,[8] Ritalta,[9] a thesis by Quillou, and a comprehensive report by Mackay[10] have been lately published. In a case of Arnheim's the viscera were also hypertrophied. For details see S. Kalischer, "Über angeborene halbseitige Hypertrophie" (*C. f. Grenz.*, 1901).

The combination of hemihypertrophy with diffuse cutaneous angioma has been described by Arnheim, Heller, and S. Kalischer.

An interesting type is reported by Wechselmann (*A. f. Dermat.*, Bd. xcvii.).

Exophthalmic Goitre

(Basedow's Disease. Graves' Disease)

Literature in the works, monographs, and treatises of Buschan, "Die Basedowsche Krankheit," Leipzig und Wien, 1894; P. Mannheim, "Der Morbus Gravesii" (sog. Morb. Based.). Gekrönte Preisschrift, Berlin, 1894; Möbius, "Die Basedowsche Kranheit," first edition, Wien, 1896; *Nothnagels Handbuch*, xxii., second edition, Wien, 1906; A. Kocher, "Über Morb. Basedowii," *Mitt. a. d. Grenzgeb.*, ix. (1902); Murray, *Lancet*, 1902 and 1905; H. Schlesinger, *W. kl. R.*, 1906; Dreyfus, *C. f. d. ges. Ther.*, xxiii. (with exhaustive discussion of the treatment); Landström, "Über Morb. Based. Eine chirurg. und anat. Studie," Stockholm, 1907 (containing references to most of the surgical papers); Eulenburg, "Die Basedowsche Krankheit," etc., *Deutsche Klinik*, vi.

Basedow was the first (in 1843) to describe this disease in Germany, although Graves, a Dublin physician, had drawn attention to the syndrome as far back as 1835.

The disease develops chiefly in middle life—in the third or fourth decade—but it has been observed much earlier and even in childhood.[11] Women are much more frequently affected than men. *Hereditary* influences can be detected in the majority of cases, although the disease is seldom directly inherited. Dejerine quotes one case in which Basedow's

[1] *B. k. W.*, 1903 (with bibliography). [2] *B. k. W.*, 1905. [3] *W. kl. W.*, 1904.
[4] "Paraffininjektion," Stuttgart, 1904. [5] *Nouv. Icon.*, xi. [6] *Z. f. N.*, xxiv.
[7] *Presse méd.*, 1903. [8] *Z. f. N.*, xxxii. [9] Abs. *R. n.*, 1906.
[10] *Br.*, 1904.

[11] Barret (*Thèse de Paris*, 1901) was able to collect forty-two cases in which it appeared before the age of fifteen. It has also been observed by, *e.g.*, Variot and Roy, in a boy of four years of age.

disease was transmitted through four generations, and Brower found it in four brothers and sisters. But as a rule there is some other nervous disease in the family, and I have very often found a predisposition for the vasomotor neuroses. The neuropathic tendency has often shown itself by signs of *nervousness* or hysteria long before the onset of the disease.

In persons with such a predisposition, exophthalmic goitre may develop without any other " direct " cause. Strong *emotional excitement* or a single great mental shock very often precede the onset, which may also follow trauma, physical exhaustion, *weakening diseases*, and difficult *labour*. It is still doubtful whether acute *infective diseases* and syphilis should be regarded as causes. I have seen it develop in a few cases after *influenza*, no other factor being detectable. In a patient of Benoit's it appeared after typhoid, and A. Kocher reports a few similar cases. Finally, it would seem that excessive use of thyroid preparations may bring on symptoms of exophthalmic goitre (see below), and that continuous use of iodine may occasionally have the same effect.

Symptomatology.—We are accustomed to recognise three cardinal symptoms in this disease, namely: 1. *tachycardia*; 2. *goitre*; and 3. *exophthalmus*, but we must remember that these are not the only symptoms.

Tachycardia is the most important. It occurs in practically every case, and in the earliest stage. It may be regarded as the initial symptom. The frequency of the pulse varies between one hundred and two hundred, and is on an average about one hundred and twenty to one hundred and forty per minute. The pulse is generally small and soft, whilst the arteries (especially the carotids) pulsate strongly and visibly. The pulse rate is specially increased by mental excitement, but the tachycardia is not solely due to increased mental excitability. Its constancy alone indicates this fact. In addition to the chronic acceleration, there are *attacks* of tachycardia and delirium cordis. The heart-sounds are loud, the apex-beat usually diffuse and slightly increased. The heart on *physical examination* often shows no abnormality. Sometimes there is a systolic murmur, especially at the base of the heart, and the heart limits are often exceeded, especially in the later stages. Symptoms of a relative mitral insufficiency have been observed in a few cases, but valvular defects are exceedingly rare, and may be regarded as complications.

Examination of the blood-pressure (Donath,[1] Spiethoff, etc.) has given varying results; I have usually found it to be increased.

It is quite exceptional for *goitre* to be entirely absent, and we should bear in mind that the thyroid may be diseased although it is not enlarged, a fact which Murray, Kocher and Hönnicke[2] have lately emphasised. The goitre usually appears after the tachycardia. It is usually soft, vascular, and pulsating, but, as Möbius has stated, and Kocher, Kraus, and others have confirmed, symptoms of exophthalmic goitre may in rare cases be associated with other forms of goitre. The degree of swelling varies greatly, and it is sometimes noticed that the gland changes in size from time to time. The superficial veins are usually dilated, and a marked *thrill* can sometimes be felt by the hand above or to the side of the thyroid. Auscultation reveals a *systolic bruit*.

Among the vascular symptoms A. Kocher includes dilatation and fragility of the vessels, which become evident during operations, and the symptom that the thyroid can be to a certain

[1] *Z. f. kl. M.*, Bd. xlviii. [2] *D. m. W.*, 1906.

degree diminished by expressing the blood from the vessels. Struma vasculosa is a constant symptom of exophthalmic goitre. If the thyroid cannnot be palpated, one should remember the so-called immersed goitre (Hönnicke).

The *exophthalmus* is, in most cases, *bilateral*. It may at first be limited to one eye, the left in particular, and may be more prominent in one eye than in the other. Sometimes it is very slight (being only recognised by those who have known the patient previously or on comparison with an earlier photograph), but it may be so marked that the eyelids cannot be fully closed, the insertions of the eye muscles are visible and the conjunctiva and cornea become the site of inflammatory processes or even of ulcers (Uhthoff, Griffith, etc.). In some rare cases, *e.g.* one described by Spalding, it has been necessary to enucleate the eyeballs.

Hascovec (*W. kl. R.*, 1906) has recently published a careful study of exophthalmus in Basedow's disease.

Complete absence of exophthalmus is not exactly rare, and as the goitre may also be absent or very slight, the diagnosis in such incompletely developed cases (formes frustes) may be difficult to establish. There are, however, a number of other symptoms which, on account of their frequency, may be regarded as typical. We shall mention them now, and analyse them later along with other less common signs. They are : *mental disturbances, nutritive disorders, motor weakness, tremor, discoloration of the skin,* and *diarrhœa.*

Let us first consider the functional symptoms other than the cardinal signs.

Ocular Symptoms.—Graefe's sign is often present. If the patient is asked to lower his eyes slowly, following the hand of the physician, the upper eyelid is not lowered along with the eyeball, or follows it only to a slight extent, and the supracorneal part of the sclera becomes visible (Fig. 429).

This sign is inconstant, and, as the communications of G. Flatau from my clinique and others show, it may occur in other diseases and occasionally in healthy persons.

There is sometimes insufficiency of the internal recti. If the patient is asked to look first at the ceiling and then at the point of his nose, only one eye assumes the position of convergence, the other deviating outwards (Möbius). This sign is only of value if there are no errors of refraction.

The palpebral aperture is frequently abnormally wide (even when there is no exophthalmus), and the patient does not wink so often as a normal person (Stellwag's sign). In one of my cases this symptom was only present in one eye. Terson[1] has also described this. On the other hand excessive winking may be seen in a few cases. In one of my patients the blepharoclonus ceased whenever he looked downwards. Widening of the palpebral aperture is regarded as the cause of Graefe's sign.

The *ocular movements* may be impaired by exophthalmus. True *paralysis of the eye muscles* is very uncommon, but even an external ophthalmoplegia has been observed (Warner, Ballet). Epiphora and lachrymation may be present (Berger[2]), and a vibrating tremor of the eyeball

[1] *R. n.*, 1903.

[2] *Arch. d'Ophthal.*, 1894, and *A. f. Aug.*, Bd. xlvi.

has occasionally been noted. Paralysis of other cranial nerves, *e.g.* the fifth and seventh, is extremely rare, but signs of such a paralysis and even bulbar symptoms have been found in exceptional cases (Jendrassik,[1] Dinkler, Bruns [2]) ; (see chapter on myasthenic paralysis).

The retinal arteries sometimes pulsate distinctly, although there are *no other ophthalmoscopic changes*. Optic atrophy has only been described in two cases, and was probably a complication, as was also the optic neuritis which I have once observed.

Mental Symptoms.—The mental condition of a patient suffering from exophthalmic goitre is hardly ever normal. He is almost always abnormally excitable, irritable, forgetful, flurried, restless, and confused. Reynolds speaks of a mental chorea. This condition is as a rule evident in the facial expression and manner of the patient. He looks timidly at the examiner, is embarrassed and restless ; his glance is unsteady, and involuntary movements betray his state of confusion. He cannot be

FIG. 428.—Woman with exophthalmic goitre. (Oppenheim.)

FIG. 429. — Graefe's sign in exophthalmic goitre. (After Bruns.)

questioned in the ordinary way ; his answers are evasive and contradictory, and there are many things which he cannot recollect, etc. etc. The degree of this disorder varies greatly in different cases and at the various stages of each case.

A marked psychosis, in the form of *melancholia*, *mania*, *hallucinatory confusion*, and excitability, or catatonia, is more rare. Atypical forms of insanity are more common. There is no specific psychosis of exophthalmic goitre. Acute delirium may develop in the last stages of severe cases, and especially those with an unfavourable course. On the other hand, certain symptoms of exophthalmic goitre may appear in the course of certain psychoses. I have seen a few cases of this kind, and Thoma [3] has described a menstrual psychosis with periodic goitre and exophthalmus.

Sleep is usually disturbed and incomplete, but in a few cases I have observed such abnormally deep sleep that the patient could not be wakened. This was associated with enuresis. Meige and Allard [4] have also recently described this symptom or attacks of lethargy.

[1] *A. f. P.*, xvii. [2] *N. C.*, 1903. [3] *Z. f. P.*, Bd. li. [4] *R. n.*, 1900.

Disturbances of the general nutrition are practically always present. The emaciation may be slight, but in some cases it may increase into marked *marasmus*, and as F. Müller [1] has shown, this may develop even although the amount of food taken is increased. Matthes found disintegration of albumen, and Magnus-Levy [2] increased excretion of carbonic acid. The great *physical weakness* of which the patient complains is partly due to this emaciation, but there is also an actual motor weakness, sometimes chiefly in the legs, which may be so great, or so much increased at certain times, that the patient collapses (Charcot). Chronic paraparesis has also been observed. Hemiplegia and monoplegia are very rare occurrences (cases of Boinet, Dinkler,[3] Maude, Ballet, etc.), and it may be that the earlier observations of this kind have been due to a combination with myasthenic paralysis. Saenger and Edinger have personally told me that they have noticed the occurrence of a periodic paralysis in exophthalmic goitre. Rosenfeld [4] has described an interesting case of acute ascending paralysis of the Landry type. Möbius has once seen paraphasia appear in the course of the disease.

True *muscular atrophy* has been noted only in a few, usually atypical cases (Ballet, Jendrássik, Miesowicz [5]). Thus, I have found almost entire absence of the gluteal muscles in a woman with exophthalmic goitre.

Tremor is almost always present, though it does not persist through the whole course of the disease. It is sometimes limited to the extremities and sometimes involves also the museles of the trunk, so that by laying a hand upon the shoulder or head we can feel a quiver of the whole body. The tremor is *rapid* and vibratory, eight to ten oscillations occurring in the second. The various oscillations are almost regular, but they may be abruptly increased. The tremor is usually aggravated by movement, and even more so by mental excitement, but it is also perceptible during rest.

The symptom was first described by Charcot (*Gaz. méd. de Paris*, 1856), and has since then been carefully studied by Marie ("Contribution à l'étude, etc., des formes frustes de la maladie de Basedow") and by Maude (*Br.*, 1892).

In a few cases there are also twitching movements like those of *chorea*.

The *deep reflexes* may be either increased or diminished, and in rare cases they may be temporarily absent.

Marked pulsation of the small arteries and capillary pulse, a double murmur at the femoral artery, and pulsation of the liver and spleen are occasional symptoms (Gerhardt).

There are numerous *trophic*, *vasomotor*, and *secretory* disorders. The skin has very often a dirty appearance, and spots of pigment, vitiligo, urticaria and other efflorescences are also often observed.[6] Pigmentation of the margins of the eyelids is specially common (Schrötter, Jellinek,[7] Teillais [8]). The discoloration may be as marked as in Addison's disease. In one case which showed extensive pigmentation of the mucous membrane

[1] *A. f. kl. M.*, Bd. li.

[2] "Handbuch d. Pathol. d. Stoffwechsels," ii., 1907. See also Clemens, *Z. f. kl. M.*, Bd. lix.; Oswald, "Der Morb. Based. im Lichte der neuren experiment. klin. Forschung.," *W. kl. R.*, 1905.

[3] *N. C.*, 1898. [4] *B. k. W.*, 1902. [5] *W. kl. W.*, 1904.

[6] Foerster's case, in which abnormal pigmentation developed in a patient with myxœdema due to absence of the thyroid, after the use of thyroid preparations, is interesting in this respect, and Kocher mentions that the pigmentation may disappear after thyroidectomy in exophthalmic goitre.

[7] *W. kl. W.*, 1904. [8] *R. n.*, 1906.

of the mouth, I diagnosed a combination of exophthalmic goitre and Addison's disease, but further experience has led me to hold strongly, even when there is marked pigmentation, to the view of an uncomplicated exophthalmic goitre, and to give the prognosis accordingly. I have several times seen the hair fall out or become grey, and occasionally show other changes of colour. Schrötter [1] found thickening of the skin from lipomatosis in one case.

Local *œdema* in unusual sites is not uncommon. It may appear for a time only, and thus show its close relationship with acute circumscribed œdema. Œdema may of course also be a result of the cardiac weakness, and in such a case it differs in no way from ordinary dropsy due to obstruction. Swelling of a myxœdematous character may also develop. The eyelids are occasionally swollen, and in a few cases which I examined this was one of the first signs of the disease. Swelling of the joints is not common, but in two cases I have seen a painful affection of the joints with muscular atrophy develop in the course of the disease. Hæmorrhage from the nose is not infrequent, and hæmorrhage from the internal organs has been described in a few rare cases. Popoff [2] has described cases in which hæmorrhage from the uterus, nose, larynx, etc., and ecchymoses were among the chief symptoms. Multiple teleangiectases were present in one case.

Hyperidrosis is the chief secretory symptom, and is absent only in a few cases. The sweating may be partial (*e.g.* in the palms of the hands, the head, etc.), or general, and it may be so excessive that the patient has to change his clothing several times during the night. *Unilateral hyperidrosis* is uncommon.

The great moisture of the skin is apparently the cause of another symptom, discovered by Vigouroux, but which is not exactly pathognomic, viz. *diminution of the electrical resistance of the skin.* This can be quickly and simply tested if we ascertain on the galvanometer the deviation of the needle with a certain number of elements, and compare this with that of a healthy person under the same conditions; it will often be found that the deviation is very much greater in a person with exopthalmic goitre. Exact investigations (Martius, Oppenheim, Kahler, etc.) have shown as follows:—

1. The absolute minimum of resistance is very low.

In order to understand this fact it must be remembered that the electrical resistance of the skin diminished under the influence of the galvanic current. When a current of medium intensity is used (of about 10 elements), the resistance is diminished to a certain degree (relative minimum of resistance); if the current is increased the resistance can be still further diminished, until finally the lowest limit is reached, beyond which it cannot be diminished by further increase of the number of elements (absolute minimum of resistance).

2. The minimum resistance is reached by the use of a relatively weak electro-motive force (25 elements as against 30-35 in a healthy person).

3. It is easily diminished by the use of weak electro-motor power.

The *secretion of urine* is often increased. It very seldom contains abnormal constituents, but albuminuria and glycosuria have both been observed. Chvostek mentions alimentary glycosuria, which has been specially noted by Hirschl (*Jahrb. f. P.*, xxii.) in severe cases, but a combination with diabetes is rare. There may also be *polydipsia*, and most patients complain of great thirst. *Amenorrhœa* is one of the uncommon symptoms, and other irregularities of menstruation and metrorrhagia may be present.

[1] *Z. f. kl. M.*, Bd. xlviii. [2] *N. C.*, 1899.

The *digestive system* is as a rule involved. Loss of appetite is as common as *bulimia*. Dryness of the mouth and pharynx are not unusual (A. Kocher). *Attacks of vomiting* occur and may develop into incontrollable sickness. *Diarrhœa* may be a very troublesome symptom. The stools are sudden and frequent, usually painless and watery, occurring four to five, and often ten or more times in the day. They contribute greatly to the patient's exhaustion. Intestinal hæmorrhages and hæmatemesis may also occur.

Respiratory disturbances are not common, but in a few cases (Notthafft, Lichtwitz-Sabrazès) they have been very distressing. Hofbauer[1] described typical respiratory disorders, both chronic and intercurrent. In the former case he found flattening of the respiration curve, retardation of inspiration and expiration, irregularities in the form and size of the various respirations, pauses in the breathing, etc. A dry, spasmodic cough is sometimes complained of. Bryson emphasised the slight degree of expansion of the thorax in inspiration, especially in severe cases, but the weakness of respiration is associated with general muscular weakness (Patrick[2]). The temperature of the body is usually normal, but in rare cases *intercurrent attacks of fever* occur (usually rise of temperature without the other signs of fever, Bertoye). A feeling of heat is often present. Headache and vertigo may occur, and there may be pain in other parts of the body, *e.g.* the ear or jaw (Kocher). Swelling of the cervical and neck glands has been noted in several cases (Gowers, Müller, etc.). Kocher says that the lymph glands in the neighbourhood of the thyroid gland are practically always hypertrophied.

Complications.—The complication with psychoses and cardiac defects has already been alluded to. The disease is most often associated with hysteria, sometimes with epilepsy, occasionally with myasthenic paralysis (Oppenheim, Brissaud-Bauer; compare p. 1036), and very rarely with tabes,[3] tetany, diabetes mellitus, and other diseases of the nervous system. Choreiform movements are not uncommon (Gowers, Dieulafoy), but true chorea is quite exceptional (Oppenheim, Sutherland). Jaundice may develop in the course of the disease. Recent observations show that exophthalmic goitre may be combined with or develop into myxœdema (Kowalewski, Sollier, Ulrich, Joffroy, Jacquemet, etc.). Gangrene and sclerodermia are rare complications (Krieger, Freund). In one case, which I had treated some years previously for exophthalmic goitre, gliosis and syringobulbia subsequently developed. Köppen and Revilliod describe osteomalacia and other changes in the osseous system, which they relate to the disease. Latzko in particular pointed to definite relations between osteomalacia and exophthalmic goitre, which he found to exist in a number of cases. Experiments relating to the connection between the thyroid and the osseous tissue are of great interest (Hanau, Steinlin, Bayon). Jaksch describes atrophic processes in the skeleton. Jacobsohn mentions the combination of exophthalmic goitre, with hemicrania and paralysis of the sympathetic, and Rosenfeld with acute ascending paralysis.

Course and Prognosis.—The disease has usually a slow onset, but it may develop acutely, especially after violent emotion. There is generally

[1] *Mitt. aus Grenzgeb.*, xi. [2] *N. Y. Med. Journ.*, 1895.

[3] Malaisé (*M. f. P.*, xxiii.) thinks this is not true tabes, but merely a sympathetic symptom.

a preceding *neurasthenic* or *hysterical stage*. Weakness, fatigue, and emaciation are frequent prodromata (A. Kocher), and chlorosis and menstrual pain sometimes precede the onset. The cardiac symptoms soon appear, followed by the other characteristic signs. Möbius is of opinion that the thyroid affection is the primary change, but that it may remain for a long time latent. The further course is a *protracted* one. The disease lasts for many years, with *remissions* and *intermissions*. It has seldom a rapidly fatal termination, but Trousseau and F. Müller have described cases in which it had an acute onset and ended fatally within one and a half to two and a half months. Mackenzie reports a case in which death is said to have occurred within three days. A few exceptional cases show that the syndrome may develop and disappear within a few days. *Recovery*, or improvement almost amounting to it, may take place within a comparatively short time.

The *prognosis* is in each case influenced by the duration of the disease, the severity of the symptoms, the general condition, and the social position of the patient. In recent, partially developed cases, with mild symptoms and only slight impairment of the general and mental condition, the prognosis is comparatively favourable. If it is possible to procure for a case of this kind the conditions of life most likely to prove helpful, we are quite justified in expecting recovery, or at least great improvement. I have observed a large number of such recoveries. In one case recovery has lasted for twenty-seven years, in another for eighteen, in four for six to eight years. Cheadle reports recovery lasting twenty, and Tissier twenty-five years. A lady who consulted me at the age of thirty-two for urticaria and other vasomotor disturbances, had had exophthalmic goitre from her fifteenth to her twentieth year, and recovered from it after an attack of typhoid. On the other hand the prognosis is very grave when the disease is longstanding and far advanced, and when the individual symptoms are very marked. The tachycardia is an important indication as to the prognosis, which becomes very grave if there is marked marasmus, dilatation and weakness of the heart, great irregularity in the heart's action, valvular disease (which one must be very cautious in diagnosing in exophthalmic goitre), much discoloration of the skin, or mental disorders. An acute development and course are also unfavourable signs, but even in such cases there may occasionally be improvement, which is sometimes very considerable and prolonged. Thus, I have seen one patient, formerly a strong young woman, drop to 75 lbs. in weight; the pulse became so rapid that it could hardly be counted, and other symptoms indicated a definite or marked development, but a change of air, dieting, and the use of galvanism was followed by great improvement, and a gain of 47 lbs. in weight. The danger to life is only great when severe disturbances of compensation have set in. Death is usually due to heart-disease, but it may result from general weakness or occur at the height of some mental disorder. Uncontrollable vomiting and profuse diarrhœa may also prove fatal.

In Murray's earlier statistics of forty cases, seven ended fatally, two remained stationary, and thirty-one improved. Of the latter, nine recovered, eight improved greatly, and fourteen showed slight improvement.

In my experience, improvement takes place more often than complete recovery. In some cases all that remains is slight *exophthalmus* or an unnoticeable swelling of the thyroid. On the other hand, in a man of

fifty, who complained of a condition of oppression, I found dilatation of the heart and tachycardia, and learned that nineteen years previously he had suffered from exophthalmic goitre with complete development of all the cardinal symptoms. In one of the most severe cases which I have ever seen, with cardiac weakness so pronounced as to have caused general dropsy, recovery took place. In another complicated case, also with disturbances of compensation, the patient, who was syphilitic, showed marked and permanent improvement after treatment with inunctions. In yet another case of medium severity, the symptoms disappeared, but they were replaced by writers' cramp and other nervous disorders. Pregnancy has an unfavourable effect upon the disease, but in several cases improvement has been observed during its course. I have once seen undoubted improvement follow jaundice. Troitzky states that in one case the disease disappeared after a strumitis erysipelatosa. In a case of Kocher's, improvement was probably due to severe nephritis with uræmia.

Differential Diagnosis.—In marked cases the condition cannot be mistaken. The exophthalmus hardly occurs, except in the intermittent form, in any other disease, except retrobulbar tumours, hydrocephalus, and aneurism. Difficulty in diagnosis therefore only arises when two of the cardinal symptoms—exophthalmus and goitre—are absent, or very slight. The diagnosis then rests upon the tachycardia and the other accessory symptoms. As many of the latter, *e.g.* the usual mental disorders, sweating, thirst, polyuria, vomiting, diarrhœa, etc., also occur in neurasthenia and hysteria, of which nervous palpitation is also a common symptom, there is some danger of our being led astray in this direction. In these conditions, however, the tachycardia is usually *not a constant* but a paroxysmal symptom.[1] The patient should therefore be frequently examined, kept for a long time under observation, examined when thoroughly at rest, etc. The other occasional symptoms, *e.g.* Graefe's symptom, pigmentation, diminution of the electrical resistance of the skin, etc., should also be looked for. Shyness, confusion, inattentiveness, and forgetfulness are more marked in the mental attitude of a patient with exophthalmic goitre than in a hysteric. But I have certainly met some cases in which I could not be sure whether the diagnosis should be one of exophthalmic goitre (forme fruste) or of neurasthenia.

The tachycardia of the climacteric may be misinterpreted in this way, and other symptoms of exophthalmic goitre may also be due to a climacteric neurosis.

Tachycardia is very seldom absent in exophthalmic goitre, but it may disappear during the stage of remission. In one case (Acchioté, *R. n.*, 1903) bradycardia is said to have occurred (?). I have seen cases in which tachycardia and loss of strength were the chief symptoms; these were always accompanied by a tendency to diarrhœa or hyperidrosis, or to diffuse or local discoloration of the skin, so that, in short, the symptoms might have been attributed either to incomplete exophthalmic goitre, to atypical Addison's disease, or to a transition form between the two. So far as I have been able to follow the cases, the course has been unfavourable. Improvement only occurred in one case, in which the condition developed in a gouty person who weighed only 80 lbs.

A *myopic eye* is not infrequently mistaken for exophthalmus. It is advisable, in doubtful cases, to compare the patient with an earlier photograph.

[1] I cannot agree with Kocher in thinking that tachycardia which occurs on the slightest occasion is more characteristic of exophthalmic goitre than the persistent form.

The symptoms produced by a *goitre pressing upon the sympathetic* are, as a rule, unilateral. Attacks of breathlessness, hoarseness, dysphagia, dilatation of the pupils, etc., are also notable signs. It is not unusual for symptoms of exophthalmic goitre to supervene upon an old goitre (Möbius). Marie, indeed, would distinguish between the "goitre Basedowifien" and true exophthalmic goitre. Mikulicz and Reinbach [1] also adopt this point of view; they speak of the "spontaneous thyroidism" of the goitre, and include slight tachycardia, tremor, vertigo, excitability, etc. As these symptoms form only a part of the symptoms of exophthalmic goitre, the condition cannot be regarded as being due to alteration in the thyroid gland.

So-called "*goitrous-heart*" has been recently carefully studied by Minnich ("Das Kropfherz," etc., Leipzig, 1904) and Kraus ("Kongr. f. innere Med.," 1906; *D. m. W.*, 1906). Kraus distinguishes two forms: 1. the cardiopathies caused by mechanical action of the goitre upon the nerves, trachea, and superior vena cava; 2. the goitrous-heart, in the narrow sense of the word, which is caused by toxins arising from the thyroid. The relation of this condition to exophthalmic goitre will be more fully discussed.

I have seen a few cases in which I could not diagnose between exophthalmic goitre or goitre with symptoms of compression. The combination of a simple goitre with nervous tachycardia may also confuse the diagnosis.

Acute iodism may produce a syndrome allied to that of exophthalmic goitre. Breuer [2] has collected a number of cases in which treatment of goitre by iodine has given rise to the symptoms of Basedow's disease.

Möbius [3] describes an "enigmatical glandular disease," some of the symptoms of which correspond to exophthalmic goitre, but instead of the goitre there is swelling of glands. The disease runs a fatal course. The glands may, however, become swollen in exophthalmic goitre.

Pathological Anatomy.—Nature of the Disease.—Examination of the nervous system has led to no uniform result. A case of Dinkler's, in which degenerative lesions were found in the brain, and changes in the roots of the cranial nerves were shown by the Marchi method, is too complicated to indicate the nature of the disease. Hæmorrhages, softenings, and inflammations have been occasionally found (Naumann, Johnstone, Lamy, Gibson, Grainger Stewart, H. White, Klien [4]; the latter has collected the results), but in some cases the pathological character of the changes is doubtful; in others there were complications, and in the rest it was doubtful whether the changes represented the cause of the syndrome; they were much more probably of a secondary nature, and due to the poison of the disease (Klien). Degenerative changes have often been found in the sympathetic, but these are inconstant and not of real importance. Ehrich,[5] who examined a sympathetic removed during life, found fatty infiltration of the ganglion cells, increase of the connective tissue between the cells, etc.; he regards these as secondary changes caused by the disturbance of circulation. Examination of the thyroid (Rénaut, Soupault, etc.) showed cirrhotic processes, hyperplasia and obliteration of the lymph-spaces. Hanau-Haenig and Ehrich describe diffuse parenchymatous hyperplasia with great structural changes of the cells and disappearance of the colloid. Ehrich also emphasises the great amount of blood and the dilatation of the vessels. Similar changes were found in

[1] *Mitt. aus d. Grenzgeb.*, vi.
[2] *W. kl. W.*, 1900.
[3] *M. m. W.*, 1905.
[4] *Z. f. N.*, xxv.
[5] *Beitr. z. klin. Chir.*, Bd. xxviii.

the glands, which to the naked eye did not appear to be altered. According to Brissaud,[1] the goitre of Basedow's disease does not differ in its pathology from ordinary goitre, and Kocher finds no specific change. See also the papers of MacCallum [2] and Humphrey.[3] It should also be pointed out that hypertrophy or abnormal persistence of the thymus often occurs in this disease (Mackenzie, Mikulicz, Hansemann,[4] Gierke [5]).

There is no lack of theories as to the nature and cause of this affection. The view that it is chiefly a *disease of the sympathetic nervous system* has much in its favour. Many of the symptoms, the tachycardia, vascular dilatation, vasomotor and secretory disturbances, etc., might be attributed to a sympathetic disease. In any case it is not right to object to this view on the ground that some of the symptoms point to a condition of irritation, others to a condition of paralysis. Such a combination is often present in nervous diseases to which no exception can be taken. The investigations of Morat, Abadie, and others show that irritation of the thoracic sympathetic may produce symptoms of tachycardia, exophthalmus, and congestive swelling of the thyroid gland. Abadie thinks the disease is due to stimulation of the vaso-dilators of the sympathetic, a view with which Dastre, Valençon, and others agree. On the other hand the vasodilators of the thyroid and the accelerators of the heart run, according to Cyon, in the *depressor* (see, however, p. 1315). François-Franck has also opposed Abadie's theory and its conclusions.

Another theory is that the disease is localised in the *medulla oblongata*. A few pathological investigations (Mendel, Kedzior-Zanietowski, and lately Klien [6]) support this view, and the experimental examinations of Filehne, who produced some symptoms of exophthalmic goitre by section of the restiform body in animals, are also in its favour. Durdufi and Bienfait, Troitzki, Tedeschi,[7] and others have come to a similar conclusion, and the theory is in harmony with the fact that ophthalmoplegia and bulbar symptoms occasionally develop in the course of the disease. It is further pointed out that in moments of great excitement, a group of physiological symptoms may appear, *e.g.* widely open eyes, protruding eyeballs, a rapidly beating heart, which in more marked degree are constant in Basedow's disease. It is possible that the pons and oblongata contain a central apparatus which, when stimulated, produces this group of symptoms (Putnam). According to Möbius, Brissaud, and others, this is a site of least resistance for the poison of exophthalmic goitre (see below). Some French writers indeed go so far as to regard Basedow's disease as a result and symptom of hysteria.

Gauthier and Möbius have lately proposed another theory, which is supported by Joffroy, Marie, Revilliod, Rehn, Jaunin, Dinkler, Bruns, Hascovec,[8] and others. It attributes the disease to *exaggerated* (Möbius), *abnormal* (Gauthier), or exaggerated and perverted (Möbius) *activity of the thyroid gland* (hyperthyroidism or dysthyreosis). This theory is based upon the fact, now recognised by every writer with the exception of H. Munk, who entirely denies it, that the thyroid is an indispensable organ, which produces substances that are absolutely necessary or have the function of neutralising toxic products of metabolism. Blum,

[1] *Arch. chir. de Bordeaux*, 1895.
[2] *Bull. Johns Hopkins Hosp.*, xvi.
[3] *Lancet*, 1905; refers to the parathyroid gland.
[4] *B. k. W.*, 1905.
[5] *M. m. W.*, 1907.
[6] *Z. f. N.*, xxv. (see literature here).
[7] *Riv. di Patol. nerv.*, 1902.
[8] *W. m. P.*, 1906; *Casop. lek.*, 1906.

and especially Cyon, have further developed this neutralising theory. According to the latter, iodine introduced through the thyroid into animal bodies is transformed into a harmless chemical substance, etc. He has, however, more recently abandoned the view of a neutralising function of the thyroid, substituting the theory that the thyroid by its secretion exercises a regulating effect upon the excitability of the nervous system of the heart and blood-vessels, an effect which is destroyed by its disease. Kishi[1] maintains that the thyroid counteracts an albuminous substance, a nucleo-proteid, which affects the blood, as a globule containing iodine is formed within the cells of the gland, which is combined with the nucleo-proteid (thyreotoxin). Blum again described his standpoint at the Kongress für innere Med., in 1906. It has also been shown experimentally that the introduction of thyroid extract, by increasing the amount of thyroid products in the animal body, will produce many of the symptoms of exophthalmic goitre (Ballet, Enriquez, Lanz). Buschan indeed found that consumption of a large quantity of thyroid tabloids had no effect upon his general health, but such an individual immunity does not prove much. Gregor also concludes from his observations on idiot children that the preparation is harmless. On the other hand Notthafft[2] describes a case of exophthalmic goitre due to excessive use of thyroid tabloids (of which a thousand were taken), and I have seen an unmarried woman develop symptoms of the disease after taking thyroid tabloids for obesity (as many as nine being taken in the day). The case was a complicated one, as signs of polyneuritis due to alcoholism had also developed. The dangers of thyroid treatment are now generally recognised, and in France, owing to the evidence of a commission appointed to examine into the matter (Franck, Potain, etc.), the drug has been placed in the list of poisons. Rehn thinks that the "exophthalmic poison" exercises a *local* effect upon the neighbouring nerves and vessels in addition to the *general* intoxication.

At all events, the theory that the disease is due to an abnormal increase in function of the thyroid gland is very attractive. We would have further to assume that the thyroid is particularly apt to acquire this morbid condition in nervous persons, and that nervous excitements are specially adapted to stimulate this organ to excessive activity. Experimental attempts to excite the secretion of the thyroid gland by stimulation of the nerves have, it is true, given negative results(Hürthle).

The view that toxic products play a part in Basedow's disease seems to receive a certain amount of support from the detection of toxic bodies in the urine of such patients (Chevalier, Boinet, and Silber). The suggestion that occlusion of the lymph spaces in the thyroid gland causes the venous blood to be inundated with the secretory products of the thyroid is also worth considering.

A few observations seem to show that an acute post-infective thyroiditis may be the starting-point of the disease (Reinhold, Breuer). Such forms of thyroiditis occur after typhoid (Gilbert, Castaigne), influenza (Gaillard), mumps (Simonin), and even independently. Quervain (*M. f. Grenzgeb.*, xv.) has fully discussed this form. A metastatic tumour of the thyroid is said to have produced the syndrome of exophthalmic goitre in a case observed by Hirschfeld (*C. f. N.*, 1906). Partial thyroidectomy may be followed by transient symptoms of the disease. Finally, in favour of the theory of Möbius, it has been noted that exophthalmic goitre sometimes develops into myxœdema (Sollier, Baldewin, Kowalewsky, Joffroy). Ehrich (*Beitr. a. kl. Chir.*, 1900) has emphatically opposed the theory of intoxication and thyroid origin, and maintains that it is a vasomotor neurosis. He endeavours to explain all the symptoms by the changes in the nervi vasorum (or their centres) and their results.

[1] *V. A.*, Bd. clxxvi. [2] *C. f. inn. Med.*, 1898.

Lemcke (*D. m. W.*, 1894) assumes that the hypothetical poison chiefly affects the muscles and makes them atonic. Askanasy (*A. f. kl. M.*, Bd. lxv.) also attaches great importance to the changes which he found in the muscles, but his muscular theory has little to support it. The hypothesis of Londe, that hypotonus of the muscles is the main symptom, and is due to an affection of the cerebellum, is still more extraordinary.

I cannot give up the view that the disease is a neurosis, but I also think that this neurosis mainly affects the thyroid gland, and in this way produces a number of symptoms. I believe, therefore, that the typical disease is localised in the central nervous system, chiefly, indeed, in the centres of the autonomic nervous system in Langley's sense (see p. 1312), and thus involves mainly the sympathetic nervous system. The altered function of the thyroid gland is therefore a secondary symptom, which in its turn gives rise to a number of other symptoms.

A similar view is held by Byrom Bramwell, Brissaud, Marie, Mikulicz-Reinbach (*Beitr. z. kl. Chir.*, 1899, and *Mitt. aus d. Grenzgeb.*, vi.), and Erb (*Med. Klinik*, 1908). Booth assumes that the central nervous system, the thyroid, and the nerves which connect these, viz., the sympathetic and vagus, may be the origin of the disease. Kocher's view is that exophthalmic goitre arises from changes of the chemical function of the thyroid, which may be due to infective, nervous, or genital influences.

A few authors (Gley,[1] Edmunds) attribute it to disease of the whole thyroid system, specially of the parathyroid glands, but recent experiments (see p. 1275) are entirely opposed to this view. We can only refer to the changes found in the central nervous system, by O. Mass, Bensen, and others, after enucleation of the thyroid changes which they interpreted as the results of intoxication.

The uncertainty which prevails as to the nature and pathogenesis of exophthalmic goitre was clearly brought out by a discussion in the American Medical Association (*Journ. Amer. Med. Assoc.*, 1905).

Treatment.—The physician's first aim should be to procure for his patient the most favourable conditions of life. He will have accomplished much if he can succeed in shutting off the source of the mental excitement. But when no such source exists, or when it cannot be suppressed, *mental and physical rest is the chief requirement.* For this reason alone it is often advisable to remove the patient from his family and occupation into another environment. *A stay in the country, in a hospital* or in *a sanatorium* may therefore be very beneficial. The physician may also contribute to the patient's mental tranquillity by his personal influence.

Moderate exercise in the open air is permissible and even necessary, but long walks and physical fatigue of any kind must be avoided. If marked tachycardia is present, the patient should remain in bed, and even in mild or moderately severe cases rest in bed in the open air may be beneficial.

Coffee, *tea*, and tobacco should be forbidden. Milk, cocoa, kefir, and acid waters should be used, and small quantities of good red wine or light beer may be given if they agree with the patient. The *diet* should be nourishing, mild, and mainly, though not exclusively, of a vegetable nature. Ziemssen has pointed out the good effect of long-continued vegetable diet. Alt (*M. m. W.*, 1906) thinks limitation of salt and fluid is the chief factor, and prefers a diet consisting of milk, whipped-cream, unsalted butter, rice, etc. Fig. 430 indicates the therapeutic results. Cod-liver oil is also recommended.

[1] *Brit. Med. Journ.*, 1901.

Sexual indulgence should be forbidden as far as possible. All exciting occupations, *e.g.* stock-exchange business, card-playing, theatre-going, should be avoided in severe cases.

Mild hydropathic treatment, especially *cold rubbings* and *lukewarm half-baths*, may have a curative effect in recent, not very severe cases. Wet packs may lessen the tachycardia. Winternitz believes greatly in hydrotherapy; he recommends, in addition to wet packs and half-baths of 24 to 22° R. (86 to 81 F.), that bottles filled with water of 8 to 10° R. should be placed along the spinal column, but Heller prefers them to be filled with hot water. The patient may be sent to the seaside, but sea-bathing is quite unsuitable.

High altitudes, especially those of about 1000 metres, such as Aussee, Triberg, St Blasien, Giessbach, etc., have often a good effect in cases which are not too advanced. Erb has lately reported improvement to have occurred in St Moritz. Mountain-climbing, however, should be abso-

FIG. 430 *a*.—Case of exophthalmic goitre before treatment. (After Alt.)

FIG. 430 *b*.—The same case as in *a*, after treatment. (After Alt.)

lutely forbidden. *A course of baths at Nauheim* is warmly recommended, and Kudowa is also said to be good. Some physicians approve of chalybeate baths (Pyrmont).

Electrotherapy has often an excellent effect. The best method is *stabile galvanisation of the sympathetic*. An electrode (the cathode) of about two to three centimetres in diameter is placed externally to the large cornu of the hyoid bone, between the angle of the lower jaw and the inner margin of the sternomastoid; another larger one is placed in the neck (about the level of the fifth to seventh cervical vertebræ). A gradually increasing current of two to three milliampères is then applied for about two to three minutes. I, along with many other physicians, have found this treatment of great service, but it must be prolonged for a considerable time. Strong currents should be avoided, as Allard and others advise, and we should remember that a few elements are often sufficient to produce a strong current. I believe that through neglect of this principle I aggravated the condition in a case which I treated at the commencement of my career as a neurologist.

Vigouroux recommends the following method of *faradic* treatment: a broad anode of seven to eight centimetres diameter is placed on the neck; a small cathode of one centimetre diameter is applied for about one and a half minutes upon the sympathetic of each side, the current being strong enough to make the sternomastoid contract; the cathode is then placed upon the motor points of the orbicularis palpebrarum muscle, the eyelids, and round the eyes; a somewhat larger cathode is next applied to the jugular, the thyroid, and the region of the heart for about ten to twelve minutes. This treatment should be carried out every second day and continued for some weeks or months. The current should be weak when applied to the heart, and strong when applied to the thyroid. *Franklinisation* (apex current to the cardiac region) is also said to have a good effect.

A few authors, such as Beck (*B. k. W.*, 1905), Stegmann (*W. kl. W.*, 1906), and Freund (*M. m. W.*, 1907), have recently approved of X-ray treatment of the thyroid, but Pfeiffer's experience (*Beitr. z. kl. Chir.*, Bd. xlviii.) is not very encouraging.

Medicinal Treatment.—Many drugs may be helpful, but none are certain in their action. *Iron, quinine*, especially the infusion of cinchona, *belladonna* (in increasing doses, Gowers), and *potassium iodide* may be tried, if general treatment is not sufficient. Iodide preparations must be used with great caution (symptoms of the disease have been seen to develop in the iodide treatment of goitre). Erb, Murray, Jacob, and others find arsenic beneficial. Kocher and Trachewski believe in sodium phosphate in doses of 2 to 10 g. (30 to 150 grains) a day. I have also found it useful. Latzko reports that phosphorus had a good effect upon both exophthalmic goitre and osteomalacia in a case of combination of these conditions. Salicylate preparations have been recommended by Chibret and Babinski, and sodium sulphanilic acid by Kirnberger. In treating the individual symptoms, *bromides*, especially potassium bromide (1·0 to 2·0 g. (15 to 30 grains) three times a day), should be given for the cardiac troubles, the anxiety and restlessness. Tschirjew has recently advocated the combined iodide and bromide treatment. *Digitalis* does not help the tachycardia, but it may somewhat lessen any disturbance of compensation. *Tincture of strophanthus* (5-8-10 drops several times a day) is of more use. Convallaria has also been recommended (Murray). Vetlesen believes in dilute sulphuric acid (ten drops three times a day). *Cold compresses*, an *ice-bag* over the heart, or the wearing of a chest-preserver may alleviate the symptoms. Opium does not affect the diarrhœa. I have found *colombo* more useful, and bromides may do good. If Möbius is right in thinking that the organism tries to throw off the poison by means of the intestine, we would not be justified in interfering with the diarrhœa. If the sweating is very profuse, *atropin* may be tried, but hydrotherapy and electrical treatment are usually more helpful.

If the exophthalmus is considerable, the patient may require to wear *glasses* to protect the eyes. Coughing, straining, and so on should be avoided as far as possible. Compression of the eyeballs has no effect. Conjunctivitis and the less common traumatic keratitis should be treated in the usual way. The exophthalmus may render *blepharorhaphy* necessary.

Modified Weir-Mitchell treatment is recommended for the marasmus.

I have never personally found *hypnotism* of any use, nor have I known of it being successful, but Möbius reports some favourable results.

"*Treatment of the nose*" has done good in a number of cases. It is always advisable to treat any swelling there may be by cauterisation or

galvano-caustic measures. Symptoms of exophthalmic goitre have, however, been produced by such nasal treatment (Schmidt, Semon). Improvement has in a few cases followed gynæcological operations.

Attempts have been made to treat the disease by the introduction of thyroid substance or extracts (thyreo-iodine, etc.), a method which is directly opposed to the theory of hyper-thyroidisation described above. The results show a small number of apparent improvements, which are difficult to explain, against a much larger number of failures. In a few cases death has been attributed to this treatment, *e.g.* by Ball.[1] As the result of my own experience I am entirely opposed to this method.

Owen, Mikulicz, Galdi, and others have given thymus (10 to 15 g. (150 to 225 grains) of fresh sheep thymus), apparently with success. Ovarian extracts have also been recommended (Delannay, Seeligmann, Moreau). In one case, in which severe exophthalmic goitre occurred simultaneously with the commencement of the menopause, I found great improvement, with a gain of 18 lbs. in weight, follow the use of this drug. Latzko and Parhon-Goldstein[2] assume that there is a connection between the internal secretion of the ovaries and the thyroid gland.

Ballet and Enriques have used serum from thyroidectomised dogs, and have found it successful. Burghart has had the same experience, and he also reports having injected the serum of a patient suffering from myxœdema into a girl with exophthalmic goitre " with decided success." Lanz and Goebel used the milk of thyroidectomised animals with the same result.

These experiments have gradually become concentrated into a positive method of treatment. Möbius[3] has instituted the method of anti-thyroid serum, made from the blood of thyroidectomised sheep, and this has had good results in his hands and in those of Schultes, Rosenfeld, Burghart-Blumenthal, Aronheim, Hempel, Lanz,[4] Dürig,[5] Christens,[6] Stransky,[7] Mayer, and others, whilst Heinze, Murray, and others have found it of no effect, and Eulenburg[8] and Rattner[9] of but little use. The " antithyreoidin Möbius," prepared by Merck from Möbius' prescriptions, is given in doses commencing at 0·5 c.cm., and increasing to 2·0, and even 5·0 c.cm., taken in wine, raspberry juice, etc. More recently Burghart and Blumenthal[10] have prepared a powder from the milk of thyroidectomised goats, called *rodagen*, the average dose of which is 75-300 grs., but which may be given in much larger doses. They and also Hudovernig have found this successful in many cases, whilst others have used it with practically no effect. Hallion and Carion have modified the method of Ballet and Enriquez, and used glycerine extract of the blood serum of thyroidectomised animals. Rogers-Beebe and Thompson[11] employ a " cytotoxic " serum of rabbits.

Surgical treatment of the goitre has been used in order to cure both the goitre and the primary disease itself (see below). *Electrolytic treatment* of the goitre has reduced it in size. Iodoform-ether—ether 20·0, iodoform 4·0, in doses of 1 c.cm.—has been injected into the parenchyma of the

[1] *Journ. Amer. Med. Assoc.*, 1905.
[2] *Arch. gén. de Méd.*, 1905.
[3] *M. m. W.*, 1903, and *loc. cit.*
[4] *M. m. W.*, 1903.
[5] *M. m. W.*, 1905.
[6] *Med. Klinik*, 1905.
[7] *W. m. P.*, 1906.
[8] *B. k. W.*, 1905.
[9] *N. C.*, 1907.
[10] *Therap. der Geb.*, 1903.
[11] *N. Y. Med. Journ.*, 1906.

gland (Pitres, Abadie-Collon). The injections should be made at intervals of some days or weeks, and continued for a long time.

Of recent years many methods, including direct excision (thyroidectomy), have been employed, with the object of rendering the thyroid innocuous.

We need not here discuss Poncet's *exothyropexia*, which has only an ephemeral interest. *Partial ablation of the goitre* was first carried out by Tillaux and Rehn, and during the last twenty years many surgeons (Rehn, Kocher, Krönlein, Mikulicz, Lemcke, Wolff, Kümmel, Horsley, Schulz, Péan, Booth, Bennecke, Majo,[1] Curtis,[2] etc.) have had very encouraging results. Marked improvement is often obtained, and not infrequently recovery.

Heydenreich's statistics show a favourable result of the operation in 82 per cent. of the cases. Starr has collected 140 cases with operation, 23 of which ended fatally, 45 improved, 3 remained unaltered, and 45 showed uncertain results. Kümmel emphatically approves of the operation, of which he reports excellent results. Some of his cases were tabulated by J. Schulz. Certainly other writers, who have had an opportunity of examining the cases upon which he has operated, have not recognised them as completely cured.

According to Sorgo's careful statistics (*C. f. Gr.*, i.), relating to 172 cases of thyroidectomy in exophthalmic goitre from 1894 to 1896, improvement occurred in 51 per cent. (marked in 15·2 per cent., and undoubted in 36 per cent.), recovery in 27·9 per cent., and failure in 6·4 per cent., whilst in 13·9 per cent. death took place during or shortly after the operation. The results are therefore, as Sorgo says, practical evidence that the operation is justifiable and useful. But it should only be undertaken when internal treatment has proved a failure, when the disease is making rapid and ominous progress, or when the individual symptoms are of excessive severity.

Reinbach has reported 18 cases from the clinic of Mikulicz: 12 were completely cured, 9 of them being examined 4 to 5 years after the operation, and 3 were markedly improved, although they were only under observation for a short time. One case only proved a failure. Although in a few cases the history of the illness does not point conclusively to the diagnosis of exophthalmic goitre, it is, on the other hand, certain that marked improvement or even recovery has followed ablation or partial removal of the thyroid in a large number of undoubted cases. The operation is always indicated when internal treatment has proved useless. Rehn, from his own practice and others, has collected 177 cases of thyroidectomy in exophthalmic goitre; of these 102 were cured, 47 improved, 4 were not improved, and 24 ended in death. He would operate whenever symptoms of pressure appear, but he also would give internal treatment the first chance, not, however, delaying the operation too long. It is very instructive to contrast these views with the statistics of Garrès' cases given by Ehrich. In these complete cure was only once obtained, and that in a case of secondary exophthalmic goitre; 2 cases showed marked improvement, though not obviously due to the operation, 1 slight improvement, and 4 were unsuccessful. These statements of Ehrich's show how cautious one must be in judging of such investigations as those of Rehn, as personal examination of these cases led him to conclusions very different from those indicated by Rehn's resumé. Ehrich entirely rejects operation on the ground of his conception of the nature and origin of the disease. The more recent reports from Garrès' clinic (Moses, *Bruns Beitr.*, Bd. lvi.), however, have a more favourable tone.

Out of 9 cases with operation, Curtis had 3 deaths. Bertha Witmer reports very favourable results from Krönlein's clinic (*Beitr. z. kl. Chir.*, Bd. xxix.).

Within recent years we have had reports and communications from Hartley (*Ann. of Surgery*, 1905), Lessing (*B. k. W.*, 1905), from König's clinic, Friedheim (*A. f. kl. Chir.*, Bd. lxxvii., and *B. k. W.*, 1905), Schultze (*Mitt. aus Grenzgeb.*, xvi.), from the clinic at Jena, Schmieden (*Therap. der Geg.*, 1907), from Bier's clinic, Czylharz (*W. m. Pr.*, 1907), Michalsky (*Beitr. z. kl. Chir.*, 1906), Landström, Klemm (*A. f. kl. Chir.*, Bd. lxxxvi.), and others, practically all expressing enthusiastic approval of surgical treatment. Kocher, with his great experience and increasingly favourable results, is the outstanding advocate of its value and efficacity. The mortality in his cases has diminished to 5 per cent., whilst he reports cure in 76 per cent. of the cases treated surgically.

[1] *Med. Rec.*, 1904.

[2] *Ann. of Surgery*, 1906.

Unpleasant post-operative symptoms, in particular psychoses, fever, vasomotor symptoms, tremor, sweating attacks of tetany, etc., certainly occur in many cases, but these rapidly disappear. The exophthalmus is the most persistent of all the symptoms of the disease.

Kocher's method consists of a combination of partial thyroidectomy—he prefers to do it in several operations—and ligature of the arteries. Preparatory treatment is advisable in order to calm the nervous system (rest, strengthening food, sodium phosphate), especially if there is marked excitement and very rapid pulse. Kocher extends the indications very widely; he advises operation in every case, at the commencement and in slight cases, whilst in severe cases he recommends *repeated* operation, etc. General anæsthesia and disinfectants should always be avoided, and hæmorrhage should be carefully arrested. It is recommended (Mayo, Bier) that the whole of the posterior capsule of the thyroid should be conserved, in order to avoid injury to the recurrent nerve and the parathyroid glands. If one operation is not sufficient, it should be repeated.

In a discussion raised in 1908 in the *Medical Clinic* (Berlin), Kocher has expressed his emphatic approval of surgical treatment (see also his statements regarding his latest statistics in *Ther. d. Geg.*, 1908); Bier agreed with him, and Erb stated that he had overcome his original aversion to surgical treatment.

To summarise our advice: surgical treatment of exophthalmic goitre, in the form of partial thyroidectomy, is a warrantable and effective method, and is specially indicated in cases where internal, hydrotherapeutic, and climatic methods have proved unsuccessful. On the other hand it should not be postponed until the terminal stages nor limited to the severest cases. It cannot, however, even yet be regarded as free of all risk, and operation constitutes a special danger to life in cases of marked cachexia and great cardiac weakness.

Finally, section of the cervical sympathetic below the upper cervical ganglion, or unilateral or bilateral resection of this ganglion, or of all three ganglia along with the section of the sympathetic to which they belong, is a method recommended by French authors (Jaboulay, Abadie, Chipault), and by Jonnesco, Schwartz, and others. Chipault, and especially Jonnesco [1] and Gérard-Marchant, Schwartz, Tomaselli, and others, report great success with this method, whilst others have had unfavourable results, rapid relapse, etc. (Peugnier), or immediate death (Deshusses [2]), and have abandoned the operation (James Berry). Cyon suggests that the depressor may sometimes have been sectioned, which would possibly explain the results (see above). According to Jonnesco, Balacescu,[3] and others, the resection must be total and bilateral, comprising the inferior cervical ganglion and the highest thoracic ganglion, which often merges into it. That would be the effective and rational operation. Among German surgeons Garrè has once performed the operation without any result, and Kocher, who has done the same, disapproves of the operation. Rehn, who has collected thirty-two cases of sympathicectomy from the literature, reports nine recoveries (28 per cent.), sixteen improvements (50 per cent.), four unchanged, and three deaths. He regards the operation as less dangerous, but much less certain than thyroidectomy. Abadie [4] has again recently spoken very warmly in favour of sympathicectomy, on the ground of his theory of the sympathetic origin of the disease and his personal experience of the treatment. Landström shows that many of the cases reported as cured by this method will not stand criticism.

We are not justified, at all events, in condemning this method of treatment. If we believe in the probable central origin of the disease, this operation would lead to interruption of the tracts by which the disease

[1] See his latest paper, abs. in *N. C.*, 1906.
[2] *Clin. Ophth.*, 1903.
[3] *A. f. klin. Chir.*, 1902, Bd. lxvii.
[4] *Congrès de l'Assoc. franc de Chir.*, 1906; *R. n.*, 1906.

is developed. But internal treatment is undoubtedly of more importance than this measure, and I have not hitherto been able to assume the responsibility for advising sympathicectomy.

Myxœdema (GULL, ORD). **Cachexie Pachydermique** (CHARCOT)

Literature: Ewald, "Die Erkrankungen der Schilddrüse, Myxödem und Kretinismus." *Nothnagels Handbuch*, Bd. xxii., Wien, 1896; Eiselsberg, "Die Krankheiten der Schilddrüse." *Deutsche Chirurgie*, 1901; Ewald, "Über Myxödem," *Deutsche Klinik*, etc., iii.; Combe, "Le Myxœdème," *Rev. méd de la Suisse*, 1897; Quincke, "Über Athyreosis im Kindesalter," *D. m. W.*, 1900; Bircher, "Fortfall und Anderung der Schilddrüsenfunktionen," etc., *Lubarsch-Ostertag*, 1896; Bircher, "Die gestörte Schilddrüsenfunktion," etc., *Lubarsch-Ostertag*, 1904; Blum, *V. A.*, Bd. clviii.; Biedl, "Innere Sekretion," *Wiener Klinik*, 1903; Magnus-Levy, *Z. f. k. M.*, Bd. lii.; Magnus-Levy, "Der Stoffwechsel bei Erkrankungen einiger Drüsen ohne Ausführungsgang," *Handbuch der Pathol. des Soffwechsels*, ii.; Pineles, *W. kl. W.*, 1902, and *Mitt. aus d. Grenzgeb.*, xiv.; Bayon, "Beitrag zur Diagnose und Lehre vom Kretinismus," Würzburg, 1903; Weygandt, "Der heutige Stand der Lehre vom Kretinismus," Halle, 1904; Weygandt, "Weitere Beiträge zur Lehre vom Kretinismus," Würzburg, 1904; Siegert, *Jahrb. f. Kind.*, 1901; Wagner-Jauregg, *W. kl. W.*, 1902, 1904, and 1907; Cerletti-Perusini, "Studi sul cretinismo endemico," Roma, 1904; Bayon, "Beitrag zur Lehre vom Kretinismus," Würzburg, 1903; Bertrand, *Thèse de Paris*, 1902; Brissaud, *Nouv. Icon.*, x., 1897; Brissaud, "L'Infantilisme vrai," *Nouv. Icon.*, xx.; Anton, "Die Formen und Ursachen des Infantilismus," *Z. f. P.*, 1906; Ferranini, *A. f. P.*, Bd. xxxviii.; Gaspero, *A. f. P.*, Bd. xliii.; Hertoghe, *Bull. de l'Acad. royale de Belgique*, 1895-99, *Nouv. Icon.*, 1899 and 1900; Hertoghe-Spiegelberg, "Die Rolle der Schilddrüse bei Stillstand und Hemmung des Wachstums," etc., München, 1900; Lévi-Rothschild, "Étude sur la physiol. du corps thyroid.," etc., Paris, 1907.

Gull and Ord called attention in the seventies to a disease which consisted chiefly in peculiar *swelling of the skin and subcutaneous tissue.*

This swelling appears first and most markedly in the *face*, then in the extremities, beginning usually in the lower and extending to the upper limbs. The face looks *swollen, pale, broad, and full.* Swelling develops round the chin and eyelids. There are only narrow slits between the greatly swollen eyelids; the nose is thick and clumsy, and the face has a heavy expression (Fig. 431). The tongue is thick and swollen.

The swelling appears also on the neck and extremities. The latter become swollen out of all shape, and the hands resemble paws. The skin is pale and cold, feels *firm* and *elastic*, but does not pit as there is no true œdema present. The *sweat-secretion* ceases, the hair and nails fall off, and the skin becomes dry and rough and scales off. At certain spots, *e.g.* above the clavicles, neck, etc., tumour-like infiltrations of the skin may appear, and infiltration of the mucous membrane, *e.g.* of the mouth and gums, with loss of the teeth, is often observed.

The patient's whole appearance is *awkward* and *ungainly*, and this impression is increased on the one hand by the *muscular* weakness, and on the other by the *diminution of intelligence* and mental dulness. Slowness of thought, speech, and movement, and an awkward waddling gait are characteristic symptoms.

The hearing is often impaired, and amblyopia may develop. Ophthalmoscopic changes (atrophy of the optic nerve, optic neuritis) are found in a few exceptional cases (Wadsworth). The other special sense impressions may also be dull.

Albuminuria, glycosuria, synovitis of the knee-joint, etc., are less common symptoms. The *temperature of the body* is usually sub-normal, and the patient generally complains of a feeling of cold.

Headache, forgetfulness, vertigo, and a *feeling of weakness* and *awkwardness* are the most marked subjective symptoms.

Anæsthesia is often present; muscular atrophy, inco-ordination, contracture, etc., are less common. The reflexes and the electrical excitability of the nerves and muscles are usually normal. Increased electrical resistance of the skin has been observed. The voice is rough, hoarse, and monotonous.

There is sometimes a marked tendency to hæmorrhage from the nose, gums, or in the skin (purpura).

The *mental changes* are as a rule characterised by simple dementia, excitability and sensory hallucinations, but *psychoses* in the form of mania, melancholia, hallucinatory paranoia, etc., have been observed in a few cases (Savage). Pilcz makes a distinction between the mental con-

FIG. 431.—Woman with myxœdema. (After Charcot.)

dition in myxœdema (slowness of thought, apathy, drowsiness, forgetfulness), and myxœdematous insanity, *i.e.* marked psychoses.

The *thyroid gland* is absent or very small, but its disappearance may be preceded by swelling of the gland.

Myxœdema affects women much more often than men (117 women to 10 men). It develops *slowly*, and has an insidiously *progressive* course, but the degree of swelling may vary greatly from time to time.

The increase in the circumference of the whole body, the swollen, broad, expressionless face, which gives a general resemblance to all the cases, the monotonous, rough voice, the thick hands and feet, like the paws of a mole, the slowness of thought and movement, and the general physical weakness make it easy to diagnose the disease, but it should always be ascertained that it is one of myxœdematous swelling, and not of œdema. The condition should not be confused with persistent œdema following chronic erysipelas or with syphilitic œdema. Dalché (abs. *C. f. Gr.*, 1902) speaks of a syphilitic pseudo-myxœdema, and of an ovarian pseudo-myxœdema of the menopause. Meige and Dide have pointed out

that similar changes of the skin may occur in dementia præcox. As regards so-called familial trophœdema, compare p. 1325.

The only result of pathological-anatomical examination to which we attach any real importance—in additon to the affection of the skin and subcutaneous tissue—is the *disease of the thyroid gland.* This organ is usually diminished in size or atrophied, a change which is constantly present (we need not discuss the changes found in rare cases, chiefly by Ponfick and Vassale, in the hypophysis). Experimental and clinical cases, some of which have already been discussed, show specially that the symptoms of myxœdema are caused by loss of the thyroid gland, and the result of the modern treatment of myxœdema is a striking proof of the correctness of this view.

Kocher and Reverdin [1] had shown that total removal of the thyroid is followed by the development of a grave morbid condition known as *cachexia strumipriva.* The patient complains of fatigue, pain and heaviness in the limbs, a feeling of cold, etc., symptoms which are followed after days, weeks, months, or sometimes even after a longer interval, by transient swelling in the face, hands, and feet. This swelling and puffiness, which present all the features described above as characteristic of myxœdema, gradually become chronic. The thoughts, speech, and movements then gradually become slower and slower. The skin is pale, cold, dry, and desquamated; the hair falls out, and the bones are backward in their growth. The patient becomes more and more feeble, and if he is not treated in the way to be described, he generally succumbs to the disease. We shall not here describe in detail the analogous symptoms produced by experiments on animals. As regards tetania strumipriva, the chapter upon tetany should be consulted.

It is a very important fact that the disease does not develop if only a part of the thyroid is removed—that is in partial thyroidectomy. It must not, of course, be forgotten, that cachexia does not always follow total excision of the gland, and that it may be very slight. This is probably due to the function of the thyroid being to some extent undertaken by the accessory glands.

Details as to the number, localisation, and nature of the parathyroid gland in man are given by Welsch, Peters, Richardson, and Alquier (see p. 1275). The part which they play in the organism and the functional disturbances caused by their loss have been studied by Vassale,[2] Gley, Jeandelize,[3] Biedl, Pineles, and Erdheim, who show that removal, disease, and diminution in function of these structures is followed, not by myxœdema, but by symptoms of tetany. See also the recent experimental work of Hagenbach, *Mitt. aus. d. Grenzgeb.*, xviii.

An observation by Seldowtisch, in which myxœdema developed in a girl of fourteen after removal of an accessory thyroid at the base of the tongue, is of great interest.

Young individuals bear the loss of the gland less well than old people.

As the symptoms of cachexia strumipriva (and thyreopriva) are identical with those of myxœdema, there can be no doubt that the chief *cause* of the disease is *want of function of the thyroid.* This gland very probably secretes a substance which has an antitoxic effect upon the toxic products of metabolism. If these products are not destroyed, they give

[1] Combe, in his prize-essay, ascribes the priority to Reverdin. Hascovec has drawn attention to the earlier contributions of Maignien to this subject, and Cooper and Dupuytren had already condemned total excision of the thyroid gland.

[2] *Riv. sper.*, 1901.

[3] "Insuff. thyreoid.," etc., Paris, abs. *N. C.*, 1903.

rise to changes in the organism which are characteristic of myxœdema. This deleterious effect is particularly felt by the central nervous system.

Recent research has also proved beyond doubt that *sporadic cretinism* is a disease identical with myxœdema, of which it is an *infantile* form (myxidiocy). The patients show the following condition (Figs. 432, 434*a*, 435*a*): dwarfism, idiocy, myxœdematous skin, unclosed large fontanelle, pug nose, protruding lips, thick tongue, hanging cheeks, defective growth of hair, half-shut eyes with swollen eyelids, short thick neck, short formless body. There are often layers of fat round the neck. A depressed position of the umbilicus is also regarded as characteristic (?). Walking is clumsy or impossible. The patient does not speak. Constipation is very common. The child develops extremely slowly: dentition is late, signs of puberty do not appear, etc. Examination by X-rays shows interesting conditions, such as defective and delayed ossification, and persistence to an advanced age of the epiphyseal cartilage (Fig. 436*a*).

Fig. 432.—Case of infantile myxœdema. (Oppenheim.)

Observations of this kind have been made by Hoffmeister, Thibierge, Gasne-Londe, Wyss, Lange, Springer, Joachimsthal, Neumann, Hertoghe, Hutinel, Kassowitz, Arguitinsky,[1] Redlich,[2] etc.

The determining factor in this disease is the absence, diminution, or atrophy of the thyroid gland, which is its cause. Kocher, Wagner,[3] Bayon,[4] and others regard this also as the cause of endemic cretinism. Others, *e.g.* Bircher, Ewald, Scholz, Kraus, Pilcz, and Pineles,[5] have, on the ground of the difference in the condition of the growth of the bones, the condition of the skin, and especially of the thyroid (absence in myxœdema, goitrous degeneration in endemic cretinism), opposed this view, whilst Magnus-Levy maintain the connection, chiefly on account of their therapeutic experience.

Pineles would distinguish between *congenital* myxœdema, due to *complete aplasia of the thyroid*, and *acquired, infantile* myxœdema. In the former the characteristic symptoms appear during the first years of life, whilst in the latter they develop between the fourth and sixth years of age, and are less marked. Kassowitz and others contradict this.

According to Argutinsky (*B. k. W.*, 1906), complete absence of the centre of ossification in the carpus indicates that the myxœdema is congenital. On the subject of cretinism consult the papers quoted at the beginning of this chapter by Weygandt, Bayon, Wagner-Jauregg, etc.

Many cases of so-called "*infantilism*" belong to this class. The symptoms are persistence of childish ways, backward growth, defective

[1] *B. k. W.*, 1906. [2] *W. kl. R.*, 1906. [3] *W. m. W.*, 1903.
[4] *N. C.*, 1904, and *loc. cit.* [5] *W. kl. R.*, 1902; *W. kl. W.*, 1902.

or rudimentary formation of the genital organs, feebleness or absence of secondary sexual characteristics (Laségue, Bourneville, Thibierge, Hertoghe, Brissaud, Meige-Allard, Ausset, Bertrand).

Brissaud, following Meige, Laségue, Lorain, and others, distinguishes two or three types of infantilism: one, myxœdematous infantilism, with symptoms of myxœdema; another, the Lorain or "anangioplasic" type of infantilism, characterised by general inhibition of growth and persistence of the infantile forms, which may be due to various causes (in particular to congenital narrowness of the vessels, absence of closure of the ductus arteriosus). Infantilism may sometimes be attributed to alcoholism in the parents and to hereditary syphilis. Mixed forms also occur (Dupré-Pagniez, Ferranini). Consult the recent papers on infantilism by Sante de Santis (Rome, 1905), Lemos (*Nouv. Icon.*, xix.), Séris (*Thèse de Paris*, 1900), Hautefeuille (*R. n.*, 1900), and especially those already mentioned by Brissaud, Gaspero, Anton, etc.

The affection described by Rummo and Ferranini as "congenital dystrophic geroderma," consisting of dryness and abnormal wrinkling of the skin, absence of hair, atrophy of the genitals, impotence, falsetto voice, pendulous belly, etc., is probably a variety of infantile myxœdema (Greco). Collari and Bueri regard it as an independent disease. These conditions seem to be allied to so-called *eunuchism*. Eiselsberg, Hofmeister, and Lanz have shown that removal of the thyroid in young animals causes atrophy of the genital organs. Bartels speaks of dystrophia adiposogenitalis.

Infantile myxœdema was formerly often mistaken for rickets, but the differences are so great that there is as a rule no difficulty now in making the diagnosis.

Among the other forms of *dwarfism* or *nanism* which might on superficial consideration be classed with infantile myxœdema, the most interesting is that of *achondroplasia*. Parrot was the first to distinguish it from rickets, and Marie gave an account of its symptoms. The disease is congenital, and is characterised chiefly by the limbs being very short, while the trunk is practically normal. The shortness affects mainly the upper arm and thigh, the fingers of the dependent arm reaching only to about the hip. In contrast to *micromely*, the skull is large and usually brachycephalic. Careful examination, especially with X-rays, shows that the diaphyses are practically normal, whilst the epiphyses are deformed and thickened, the interarticular cartilages being prematurely ossified and irregularly formed. The ends of the long bones, especially the humerus, cannot therefore reach the sockets, etc. The typical symptoms include the "main en trident" (Marie), in which the fingers are almost the same length, and stand apart like the prongs of a trident. In contrast to congenital myxœdema, the intelligence is usually unimpaired, the skin unaffected, etc.

Observations of this kind have been published by Cestan, Apert, Buck, Mery, Vilaire-Cabèche (*Thèse de Paris*, 1902), Debove, Parhon Durante (*Nouv. Icon.*, 1905), Comby, Lannois-Apert, etc. I have seen a case which was specially interesting because the parents of the child were related to each other, and a tendency to dwarfism was apparent in other members of the family. Leriche describes a familial form (*Gas. des hôp.*, 1903). A comprehensive description is given by Porok-Durante (*Nouv. Icon.*, 1905), and a resumé by Schirmer in the *C. f. Gr.*, 1907.

It is doubtful whether achondroplasia can be distinguished from fœtal rickets or from *chondrodystrophia fœtalis hyperplastica* (Kaufmann, Johannssen, etc.). Virchow's case, upon which he founds his theory of cretinism, rightly disputed by Schmidt and Weygandt (*N. C.*, 1904), belongs to this class.

See also on this subject Berger (*Fortschr. d. Röntgenol*, xi.), Dieterle (*V. A.*, Bd. clxxxiv.), Porok-Durante.

Patel describes a case of dwarfism limited to the lower extremities and associated with congenital dislocation of the hip-joint.

So-called *mongolism* has much in common with infantile myxœdema, *e.g.* absence of closure of the large fontanelle, retarded dentition, protrusion of the abdomen, umbilical hernia, etc., but the longitudinal growth is very slightly, if at all inhibited, and there is occasionally even a tendency to partial giantism; the mental condition is in the later stages characterised rather by violence and restlessness, the growth of hair is not affected, and thyroid treatment has no effect (Kassowitz), at least as regards the idiocy. Among the other symptoms are smallness of the skull, the head being very low; oblique, slit-like palpebral apertures, epicanthus, and lingua scrotalis, the flaccidity of the joints, the cheerful character of the imbecility, the atrophy of the terminal phalanx, and the shortening of the middle phalanx of the little finger revealed by

X-ray examination, etc. We would refer to the papers by Thiemich (*M. f. Kind.*, 1903), Weygandt (*N. C.*, 1905), Siegert (*B. k. W.*, 1906), Neurath (*W. m. W.*, 1907), Schüller (*W. kl. W.*, 1907), Fujisawa (*Jahrb. f. Kind.*, Bd. lxii.), and Vogt (*N. C.*, 1906).

Giantism (see following chapter) may be associated with infantilism, as shown by the cases of Brissaud-Meige, Launois-Roy, Redlich, etc.

Attempts have been made during the last few years to relate other diseases and disorders of nutrition to the absence of defective function of the thyroid. We have already referred to the theory of tetania strumipriva, and to the hypothetical relation of sclerodermia, etc., to the thyroid. Putnam has stated that many disturbances of growth and nutrition may be explained by defective, insufficient function of this organ. Quincke, under the name of " infantile arthyreosis," describes conditions of mental under-development with trophic changes in the teeth, etc., which differ from cretinism in the normal growth of the body and the later date at which it appears. He advises that the possibility of disease or hypofunction of the thyroid gland should be considered in every form of imbecility. Heubner, O. Müller, and others, give the same advice.

Hertoghe (*Nouv. Icon.*, 1899) goes furthest in this respect. He maintains that there are very numerous intermediate stages between marked myxœdema and the normal condition, which he calls " benign hypothyroidea " or " abortive myxœdema " Although it is probable that there are many degrees and forms of functional disturbances of the thyroid, and slight and indefinite symptoms corresponding to these, Hertoghe's contention loses weight from the fact that there is hardly a symptom or syndrome which he does not include under this heading of defective function of the thyroid gland. Adenoids, hypertrophy of the mucous membrane of the nasopharynx, abnormal fragility of the bones, defective nutrition of the teeth and hair, uterine diseases of various kinds, metrorrhagia, the formation of varices, constipation, etc., and lately also rickets have all been ascribed to the thyroid. Although we very much doubt this view, we are led by these theories to consider that the condition of the *thyroid* is of great importance, not only in cases of fully developed myxœdema and cretinism, but in those where only some of these symptoms are present.

So-called *adiposis dolorosa* or Dercum's disease cannot be confused with myxœdema after careful examination. This condition, described by Dercum (*Univ. Med. Mag.*, 1888; *Amer. Journ. Med. Sc.*, 1902), is chiefly characterised by an accumulation of fatty masses on the surface of the body. These are distributed in a general, diffuse, or disseminated manner. In the latter case there is usually an asymmetrical development of lipomata. The diffuse and the circumscribed form may be combined. The face, hands, and feet are usually unaffected. The second characteristic is that pressure of the fatty mass produces *pain*, sometimes very acute, sometimes slight. There is often also spontaneous pain, especially on over-exertion. Spiller found that the nodules become harder and larger during the attacks of pain, possibly from congestion of the vessels (Weiss,[1] Schlesinger, Alsberg). *General weakness* or *asthenia, and mental changes* are the most common accessory symptoms, and there are occasionally sensory, vasomotor, and trophic disturbances (articular affections, sclerodermic changes in the skin, etc.). I have seen a case in which pressure on the skin led to diffuse hæmorrhage (sugillation), and trophic disturbances appeared in the nails. Sezary (*Rev. de Méd.*, 1907) also noted sugillation. A complication with epilepsy, retinitis, etc., has been described by Dercum. Hysteria may also occur (Oppenheim). The disease very specially affects *women*: among 27 cases Weiss found only 4 male patients. A relation to the menopause is often noticed. A few cases examined post-mortem suggest that an affection of the *thyroid* gland is the cause of the syndrome (Dercum, Burr, Henry). More recently Dercum, in a case examined along with M'Carthy, found changes in the hypophysis, and new-formed lymph glands in the fatty nodules, whilst the thyroid was normal. Cases of adiposis dolorosa have been described since by Dercum, by White, Eshner, Collins, Roux-Vitaut, Féré, Achard-Laubry, Rénon-Heitz (*R. n.*, 1903), Strübing, Oddo-Chassy, Ballet (*Presse méd.*, 1903), Debove (*Arch. gén. de Méd.*, 1903), Pennato (*Rif. med.*, 1903), Carnot (*R. n.*, 1906), Prunier (*Nouv. Icon.*, xx.), Guillain-Alquier (*Arch. de méd. Expér.*, 1906), and many others. A resumé has been

[1] *B. kl. W.*, 1903.

published by Weiss in the *C. f. Gr.*, 1904. Strübing states that slight forms of the disease are common. I have also seen a few typical cases, but I am opposed to a wider application of the term, as simple adiposis is often associated with nervous disturbances or due to the neuropathic diathesis.

It is not probable that there is any relation between adiposis dolorosa and " multiple symmetrical lipomatosis " or adenolipomatosis, especially as the latter chiefly affects the male sex. We cannot discuss the various theories advanced, *e.g.* by Grosch, Askanazy, Köttnitz, Alsberg, etc., but we might mention that Curling and Madelung would relate it also to the thyroid gland. We would refer to the papers by Verneuil-Panas, Israel, Hayem, Labbé-Ferrand, Launois-Bensaude, Sinnhuber, and Rothmann as valuable contributions to the subject.

Destruction of the glandular tissue within the thyroid gland (*athyreosis*) is the chief cause of myxœdema. So far we do not know what process causes this atrophy. Its result is a *hyperplasia of the connective tissue* in the skin and an accumulation of *mucin* in the tissue (Halliburton, Thierfelder), but it is not certain that the swelling of the skin is caused by the increase of mucin in the tissue.

Among the *causes*, chill, emotion, trauma, and difficult labour have been mentioned. *Heredity* should not be underestimated. Syphilis is

a *b*

FIG. 433, *a* and *b*.—Result of thyroidin treatment in myxœdema. *a*, Swelling of face before treatment. *b*. Face after several weeks' use of thyroidin tabloids. (Oppenheim.)

said to have been the cause in a few cases, but its etiological importance is still doubtful. French writers talk of a syphilitic pseudo-myxœdema (see above). Hoche has seen it follow carbonic-oxide poisoning. Burghardt mentions a case in which it developed after removal of the thyroid which had undergone actinomycotic degeneration. Some cases exist which show that sporadic cretinism may follow an acute thyroiditis in early childhood (Shield). Ceni and Agostini [1] suggest that maize-poisoning in the parents may cause malformation of the thyroid in the descendants.

The *prognosis* is on the whole unfavourable, although marked improvement, possibly recovery, has occurred spontaneously in a few cases. We have no definite knowledge of the results of *treatment*, but some of those obtained justify great hope as to the future.

Treatment.—Schiff was the first to show, by his experiments on animals, that removal of the thyroid did not produce the usual results if the thyroid of an animal of an allied species were implanted in the abdominal cavity in such a way that it remained capable of function. Upon this discovery, Bircher, Lannelongue, Horsley, and others have built up the modern treatment of myxœdema. At first thyroid tissue from anthropoid apes, sheep, calves, etc., was used ; subsequently an *extract of thyroid gland* was

[1] *Riv. di Patol. nerv.*, 1902.

FIG. 434*a*.—(After Railton-Smith.) Cretins before thyroidin treatment.

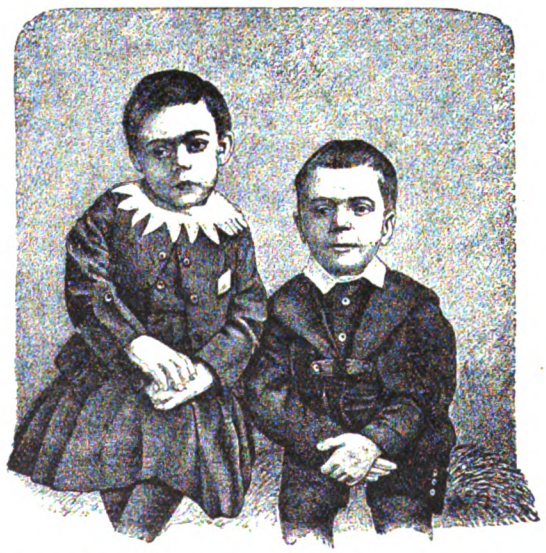

FIG. 434*b*.—(After Railton-Smith.) Cretins after thyroidin treatment.

injected subcutaneously (Murray, Bouchard, etc.), and finally, the thyroid tissue itself, or an extract prepared from it, was given *internally* (Howitz, Vermehren, Fox, Mackenzie, etc.). The *thyroidin*, which is the active substance, is contained in a glycerine extract, and is obtained from this. Baumann's discovery that the thyroid contains an organic iodine compound, viz. *thyroidin*, which forms an active constituent of the gland, is a very important one. But neither this preparation nor any other, *e.g.* thyreaden, etc., can be as efficient as the whole organ (Gottlieb, Drechsel, Stabel, Wormser, etc.).

If the gland be administered in substance, we should commence with a

FIG. 435*a*.—Infantile myxœdema before thyroidin treatment.

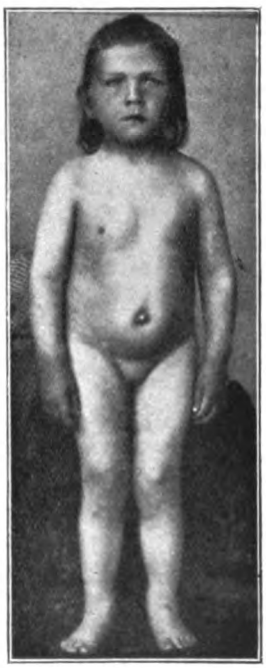

FIG. 435*b*.—After thyroidin treatment.

(After Alt.)

fraction of a gramme, gradually increasing it to 1 to 2 g. (15 to 30 grains). Some authors recommend larger quantities, given, not daily, but twice a week. Tabloids made of dried, compressed gland substance, by Burroughs, Wellcome & Co., should be chiefly used. Ewald regards these as wholly reliable. Portions of a tabloid ($\frac{1}{4}$ to $\frac{1}{2}$) should at first be given, the dose being increased according to the need and result up to five tabloids per day. Fig. 433 shows the effect that may in a short time be obtained by this treatment. Merck prepares a thyreoidinum siccatum, of which 0·1 to 0·2 may be given for each dose (in pills or tabloids). Other preparations have been recommended by Leichtenstern, Lanz, and others. The latter regards aiodin as a rational and effectual product of thyroid gland. Cunningham and also Buchanan approve of the thyreocolloid of

the fresh thyroid substance. Notkin thinks thyreoidinum depuratum is the effective substance.

During this treatment the patient should be kept mainly upon a vegetable diet. The treatment requires very careful supervision. Although it is true that healthy persons can sometimes take large quantities of thyroid substance without any ill effect (Buschan, Becker), as a rule it produces symptoms of poisoning, viz. tachycardia, dyspnœa, anorexia,

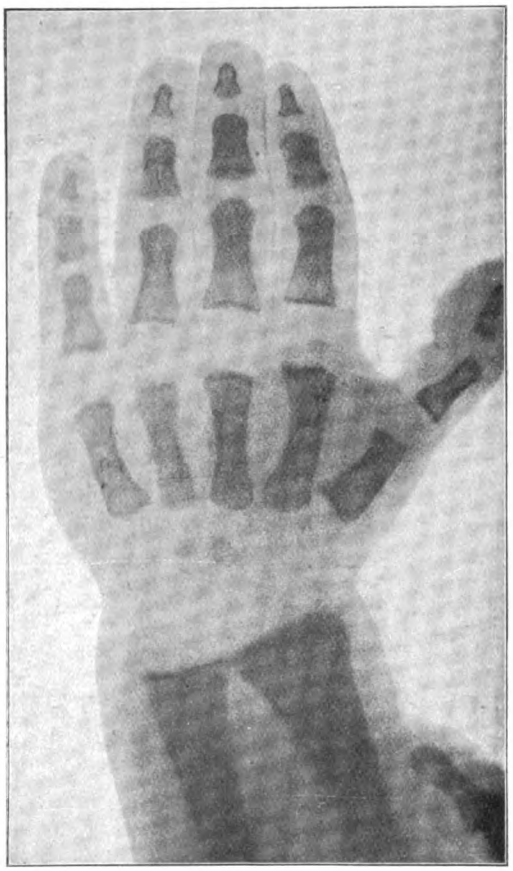

FIG. 436*a*.—Skiagram of hand in a girl aged thirteen with infantile myxœdema. (Case of W. König and Oppenheim.)

emaciation, etc., as has been mentioned in the foregoing chapter. On this subject consult Magnus-Levy (*loc. cit.*).

The experience of Murray, Marie, Haskovec, Beclère, Pilcz, and others shows that the use of thyroid preparations in myxœdema often gives rise, especially immediately after their administration, to palpitation, tachypnœa, thirst, loss of strength, albuminuria, and even uræmic symptoms and glycosuria (Ewald). In one case death is said to have followed a small dose, and in another the myxœdema developed into exophthalmic goitre. There is, however, no doubt that in some of these cases the symptoms have been caused by the use of decomposed thyroid substance

(Lanz, Stabel, Cunningham). The treatment requires to be carried out with special care in infantile myxœdema (Neumann, Siegert). Bédart and Mabille, with whom Ewald agrees, maintain that the dangers of thyroid treatment may be diminished by simultaneous administration of arsenic.

Transplantation of healthy thyroid glands has been again adopted during recent years. Favourable results are reported by Christiani (*Semaine méd.*, 1905), Kocher, Kummer-Gauthier

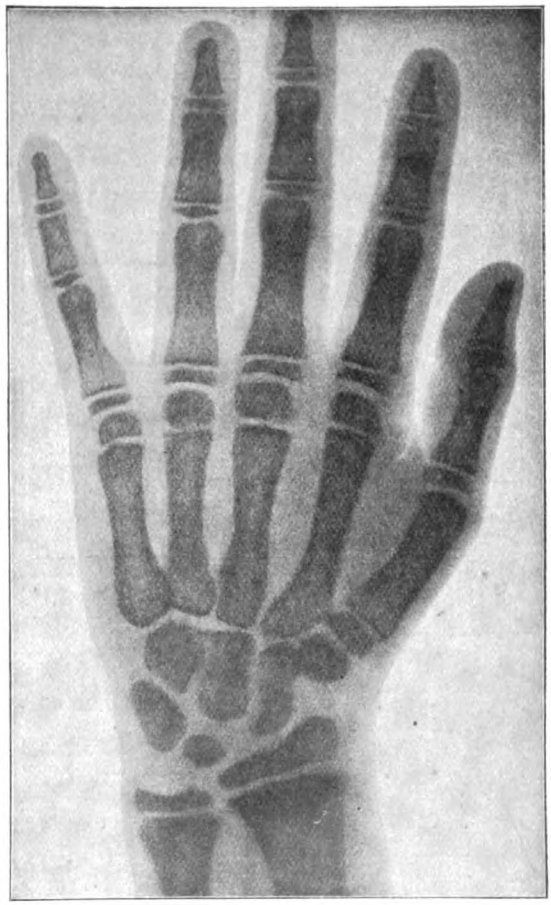

FIG. 436*b*.—The same hand as in 436*a* after a year's thyroidin treatment.

(*Acad. de Méd*, 1905). Payr (*D. m. W.*, 1906) has transplanted into the spleen of a myxœdematous child, a portion of its mother's thyroid gland, with marked success. See also Payr, *B. k. W.*, 1908.

The result of thyroid treatment of myxœdema may, on the whole, be regarded as certain and excellent. As a rule the result is not achieved by a single course of treatment, as its interruption is followed by relapses, which require it to be once more resumed.

Warmth has a beneficial effect upon the condition of the child, and it is therefore advisable for it to spend the most severe part of the year in a southern climate.

Kocher and Leichtenstern have treated cachexia strumipriva in the manner just described. Leichtenstern first used subcutaneous injections, but since then he has given fresh thyroid substance internally (on an average 75-150 grs. per week, gradually increasing the dose). The result was *excellent*.

Thyroid treatment has also proved of value in sporadic cretinism, as shown by the observations of Murray, Carmichael, Bramwell, Osler, Thomson, Koplik, Bourneville, Sklarek, H. Neumann, Ewald, Heubner, Hertoghe, Heller, Bézy, Necas, Ausset, Bruggen, Russow, Barbour, Heyn,[1] Bourneville-Lemaire,[2] Wendenburg,[3] and others. I have also to report some very satisfactory results. The marvellous effects of thyroid tabloids is shown by Fig. 434, *a* and *b*, which represent two cretin brothers before and after the treatment. The case was reported by Railton and Smith, and reproduced by Ewald. See also Fig. 435. In this case there was immense improvement, although not recovery. The influence of the treatment upon the mental development has been specially noted (Siegert, etc.). In our experience this has been considerable.

It is a very interesting fact that the effect of this treatment extends to the process of ossification, and that in a comparatively short time the backward bone formation may be made up under its influence (Gasne-Londe, Springer, Oppenheim, etc.; compare Fig. 436, *a* and *b*.).

As regards endemic cretinism, the statements differ. Régis, Wagner-Jauregg,[4] Magnus Levy, Weygandt, and others have found thyroid treatment successful, whilst Scholz and others have found it to have no effect.

Thyroid treatment has also been recommended for *adipositas dolorosa*. It has entirely failed in two of my cases. Türk recommends inunction with Crédé's ointment (abs. *B. k. W.*, 1904), but his observation is not convincing.

Acromegaly (Marie), Pachyakria (Recklinghausen)

Literature: Marie, *Rev. de Méd.*, 1886, *Nouv. Icon.*, 1888 and 1889, *Prog. méd.*, 1889, *Rev. de Méd.*, 1890; Sternberg, "Die Akromegalie," in *Nothnagels Handbuch*, vii., 1897 (containing good bibliography); Erb, *A. f. kl. M.*, Bd. lii.; Marie-Marinesco, *Arch. de méd. expériment.*, 1901; Eulenburg, *Realenzyklopädie*, i., 1894; Schultze, *Z. f. N.*, xi.; Strümpell, *Z. f. N.*, xi.; Arnold, *V. A.*, Bd. cxxxv.; Fraenkel-Stadelmann-Benda, *D. m. W.*, 1901; Benda, *Deutsche Klinik*, etc., iii.; Josefson, "Studier öfver Akromegali," etc., Stockholm, 1903; Eiselsberg-Frankl-Hochwart, *W. m. W.*, 1907; Frankl-Hochwart, *Z. f. N.*, xxxiv.

This disease, with which we have been more familiar since Marie described it in 1886, may occur at any age, but is most common in youth and middle life. Men and women are almost equally liable to it. Trauma and great emotion are regarded as causes. Heredity or a neuropathic family tendency seems to play a certain, though subordinate part, and direct heredity has been ascertained in a few cases (Schwoner, Bonardi, Cyon; the latter has seen it in three children of one family). Lead-poisoning, infective diseases, labour, etc., are given as the causes in exceptional cases, but they may have acted merely as the exciting factors. The disease almost always develops *insidiously*, beginning as a rule with dragging pains and paræsthesiæ in the limbs along with a feeling of general weakness. In women cessation of the menses is sometimes one of the earliest symptoms.

The characteristic feature of the disease is *enlargement or thickening*

[1] *A. f. P.*, Bd. xli. [2] *Progrès méd.*, 1904. [3] *M. f. P.*, xx. [4] *W. kl. W.*, 1904 and 1907.

of the distal ends of the limbs, and of the nose, lips, lower jaw, etc. *The enormous development of the hands and feet* is very striking, the fingers and toes being specially affected.

The swelling involves the bones and the soft parts, so that the hand is enlarged *in toto*, is thickened and clumsy (though not deformed). The thick, swollen, and sometimes lengthened fingers resemble sausages attached to the hand (compare Figs. 438 and 439). Marie distinguishes between the long-handed and the broad-handed types. The skin is thick, moist, spongy, and swollen. The thickening may also involve the bones of the wrist and ankle-joints, whilst the metacarpi and metatarsi are not usually greatly affected. The long bones of the extremities are not markedly involved, so that the enormous hand may be attached to a

FIG. 437.—(After Schulze.) Shape of the head in acromegaly.

slender forearm. The spinal column, clavicle, and sternum are generally affected. Cyphosis of the inferior cervical and superior dorsal vertebræ is the most constant spinal change, and the ends of the spinous processes are also often thickened. The sternum is broadened and thickened *in toto*, but the ensiform process is specially prominent, the sternum forming a double projection (Sternberg). Thickening of the manubrium sterni may give rise to diminished resonance, which Erb attributes to the thymus. The ends of the clavicles are thickened and raised, and the ribs and patella are massive.

In the head (Figs. 437, 438, and 439) the facial bones are most affected. The external occipital protuberance may also be very prominent (Schultze), and the circumference of the skull greatly increased. The condition of the lower jaw is specially characteristic ; it projects beyond the upper jaw, especially in the middle line (prognathism), the chin being very prominent and its alveolar process sometimes encircling the upper jaw. The

zygmotic arch is also very protruding. The bones and soft parts of the face and head are hypertrophied. The nose is swollen, thick, and turned-up ; the lips are heavy (Figs. 438, 439), the tongue broad, thick, elongated, and possibly of enormous size. Hypertrophy of the mucous membranes is common. The larynx is often enlarged in every part, the mucous membrane thickened, and the voice hoarse and deep. The lymphatic glands of the neck may be swollen.

Goitre is sometimes present, but the thyroid is more often diminished in size.

The thickened, bloated skin has in rare cases a myxœdematous appearance. Fibroma, neuroma, and keloids (Schultze) are sometimes

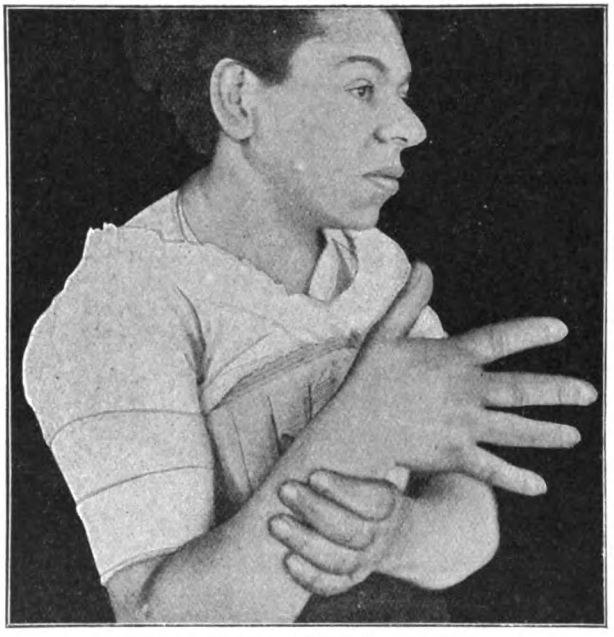

FIG. 438.—Acromegaly. (Oppenheim.)

found. The hair is not usually changed, but in one case it fell out and in another the individual hairs became thickened (Parhon, Goldstein).

Graves lays stress upon the formation of gaps between the teeth (*M. f. P.*, xvi.).

Vasomotor disturbances may develop in the course of the disease (Chvostek,[1] Bonardi, etc.). Böttiger,[2] observed Raynaud's symptoms in one case. We have already referred to the supposed relations of sclerodermia to the hypophysis. The internal organs may also become hypertrophied and enlarged (*splanchnomegaly*). The heart, in particular, tends to be affected, and functional disturbances are frequent (Huchard, Fournier, Bonardi, Chvostek, etc.). The vascular system is often involved.

A condition resembling arthritis deformans is sometimes present in the

[1] *W. kl. W.*, 1899.

[2] *M. m. W.*, 1899.

joints. A form of joint affection allied to intermittent articular dropsy has been observed by Chvostek.

The patient's whole habit of body sometimes corresponds to the enlargement of the various parts, and not a small number of acromegalics are *giants* in size.

While these anomalies slowly develop in the course of years, the patient's *general health* usually suffers. The movements become heavy and awkward; the patient is *apathetic, lacking in energy,* and *drowsy.* Marked *psychic disturbances* may occur. Joffroy and Farnarier describe dementia, and Garnier acute mania. A combination with epilepsy has been noted in several cases. *Headache* is very common, and other symptoms of a *cerebral tumour*—chiefly of a basal tumour—may occur in acromegaly, although they are by no means constant. These include, according to the observations of Hertel,[1] Uhthoff,[2] Strümpell, Strzeminski,

FIG. 439.—Acromegaly. (Oppenheim.)

Mendel,[3] Finzi, Josefson, and others, *atrophy of the optic nerve, optic neuritis,* and especially visual disturbances of the type of bitemporal homonymous *hemianopsia,* hemiachromatopsia, unilateral blindness with hemianopsia of the other eye, etc., and also *paralysis of the ocular muscles* and *hemianopic immobility of the pupils* (compare with chapter on brain tumour, p. 912). Exophthalmus and various disturbances of the trigeminal nerve (neuralgia, hyperæsthesia, anæsthesia), were occasionally present.

Bulimia, polydipsia, and *polyuria* are common symptoms. *Glycosuria* and true diabetes are not unusual. In the symptomatic form of diabetes the sugar is secreted in a somewhat spasmodic manner, and is influenced by diet (W. Schlesinger[4]). Strümpell and Chvostek mention alimentary glycosuria; the latter saw in addition in one case paroxysmal hæmoglobinuria occur in the course of the disease.

[1] *A. f. Ophthal.,* 1895.
[2] *B. k. W.,* 1897; *D. m. W.,* 1901; "Bericht der 34. Ophthalm. Gesellsch.," Wiesbaden, 1908.
[3] *B. k. W.,* 1895 and 1900.
[4] *W. kl. W.,* 1902.

The movements of the trunk and limbs are not as a rule greatly impaired, although a certain heaviness and weakness become evident. Localised muscular atrophy has been observed in a few rare cases, *e.g.* by Duchesneau, Bregmann, and Huismans.

The tendon reflexes are normal or exaggerated, or they may be absent (Mendel).

Pain is said by Souza-Leite [1] to be a rare occurrence, but on the other hand Sainton and State,[2] who have specially studied this question, regard it as very frequent, indeed as being present in half the cases. It occurs in the spinal column, the extremities, which it may affect symmetrically, and in the viscera. These authors distinguish between osteoarticular, neuralgic, muscular, tabetic (?), and acroparæsthetic pain, and attribute it to the osseous process and the neuritic, spinal, and root affections.

Paræsthesia, especially in the hands, is a typical symptom. Objective disturbances of the sensibility are not uncommon, and I have once found an isolated thermo-anæsthesia.

The disease as a rule has a *chronic* course, and may last for many years, but an acute onset and rapid course have occasionally been observed. Remissions sometimes take place. The patient succumbs to marasmus, diabetes, heart-disease, or to the symptoms caused by cerebral tumour.

Complications frequently occur. Some of these, such as epilepsy and diabetes, have been mentioned among the symptoms, but they are often merely complicating diseases. This is the case also as regards the rare cases of localised paralysis and atrophy (Duchesneau, Bregmann, Huismans,[3] Bonardi). Bonardi mentions a combination with tabes, and Petrén,[4] with syringomyelia. Association with myxœdema and exophthalmic goitre has also been noted. I have once seen an incompletely developed form combined with myoclonic dementia.

Pathological examination reveals many changes. Some of these can be detected with almost anatomical distinctness by the use of X-rays.

The bones show partly actual thickening and deformities due to projection of certain parts, partly deepening of the vessel spaces and increase of the roughness at the insertions of the tendons and ligaments. Although there is no massive, stalactite-like formation of osteophytes, small exostoses, are found often in large numbers, especially on the skull and vertebral bodies. The muscular insertions are often very prominent, and the air sinuses are usually dilated. As a rule the hypertrophy of the bones is most marked in the lower jaw.

H. Curschmann (*Fortschr. d. Röntgen.*, ix.) describes atrophic processes in the bones.

The acromegalic skull is characterised by unequal thickening of the bones, deepening of the frontal sinus, and very specially by deepening and bulging of the sella turcica. I was the first to show that the latter change could be demonstrated in the living patient by the use of the X-rays (compare Fig. 342*a* and *b* on Plate VIII. in the chapter on brain tumour), and my observation has been confirmed by Fuchs, Launois-Roy (*Nouv. Icon.*, xvi.), Josefson and Béclère (*Soc. méd. des hôp. de Paris*, 1902; *Pr. m.*, 1903), Erdheim (*Sitz. d. k. A. d. W.*, Wien, Bd. cxiii.), Schüller-Robinson (*W. kl. R.*, 1904), and many others. See also Schüller: "Die Schädelbasis im Röntgenbilde," *Fortschr. auf dem Gebiet. d. Rönt.*, Ergänz. ii., 1905, and Fürnrohr, "Die Röntgenstrahlen im Dienste d. Neurol.," Berlin, 1906. On the other hand Marie, Launois-Roy, and Béclère have demonstrated other peculiarities of the bones in acromegaly by the X-rays.

Careful examination of the affected bones reveals thickening of the periosteum, new formation of periosteal bone, thickening of the tendinous and aponeurotic insertions. Conditions resembling arthritis deformans

[1] *Thèse de Paris*, 1890.
[2] "La forme douloureuse de l'Acromégalie," Paris, 1900.
[3] *Therap. der Geg.*, 1903.
[4] *V. A.*, Bd. 1900.

are present in the joints. The skin above the thickened parts is also thickened and hypertrophied, and there is hypertrophy of the connective tissue in the glands, vessels, and nerve sheaths (Marie, Marinesco, Arnold). The spinal ganglia and the sympathetic have occasionally been hypertrophied.

The brain, eyeballs, and abdominal organs may also be enlarged. Asymmetrical degeneration of the posterior columns and also rarely of the lateral columns was found in a few cases (Arnold, Dallemagne, Bonardi). The *thymus* is often, though not always, *persistent*, and sometimes hyperplastic.

The *thyroid glands* have usually been found to be diseased, and are either small and atrophied or show *goitrous degeneration*. In a few cases they are simply hypertrophied.

An affection of the hypophysis is the most constant change (Marie). It is usually hypertrophied and swollen, and tumours of the type of *adenoma*, pituitary goitre, sarcoma, and glioma have been described.

Percy Furnivall found enlargement of the hypophysis in thirty-one out of thirty-four cases; in five of these there was simple hypertrophy, in one vascular hypertrophy, in one hypertrophy of the posterior lobe with colloid degeneration, in one hypertrophy of the anterior lobe, in one colloid degeneration with hæmorrhage, in six adenoma, in four sarcoma, in one psammosarcoma, in one glioma, in one glioma and sarcoma, and in three tumours of indefinite character, etc. In only three cases out of the thirty-four was the hypophysis not hypertrophied. In one of these the gland resembled a soft adenoma, in the second there was necrosis with softening, and in the third fibrous degeneration. Out of twenty-four cases in which the thyroid was examined, it was found enlarged in eleven, enlarged and degenerated in three, etc., and normal only in five. The author concludes that the hypophysis is hypertrophied in every case of acromegaly.

See also Woods Hutchinson (*N. Y. Med. Journ.*, 1900), Benda (*B. k. W.*, 1900), Cagnetto (*V. A.*, Bd. clxxvi.), Josefson, Stadelmann (*Z. f. k. M.*, Bd. lv.), Collina (*Riv. di Pat.*, 1903).

The hypertrophy is sometimes absent, microscopical examination showing changes of the type of cell hyperplasia, *e.g.* the cases described by D. Lewis (*Trans. Chicago Soc.*, 1904), Widal-Roy-Froin (*Rev. de Méd.*, 1906). In a few cases the hyperplasia also extends to the hypophysis, thyroids, and parathyroids (Ballet-Lavastine, *R. n.*, 1904).

The hypertrophy of the hypophysis causes compression of the adjacent parts of the brain and cranial nerves, and erosion of the sella turcica and sphenoid bone, sometimes to such an extent that the tumour projects below the pharyngeal mucous membrane.

The *diagnosis* can only be made without difficulty in advanced cases, whilst in the initial stage, in which there are practically only subjective symptoms, it may prove very perplexing. A satisfactory change has, however, taken place in this respect since I have been able to demonstrate the enlargement of the sella turcica in the living patient. The diagnosis of acromegaly has thus acquired a sure foundation, and, thanks to this fact, we have during recent years gained some knowledge of the initial stages and the *atypical* forms of the disease, especially of those in which *general adiposity* and *amenorrhœa*, or diminution of sexual functions, form the main feature in the symptomatology (see p. 912, referring to Fröhlich, Axenfeld, Erdheim, Bartels, Uhthoff, etc.). I have during the last few years seen a number of such cases, in which acromegaly, in the strict sense of the term, was entirely absent, or was indicated merely by enlargement of the nose, and so on.

As acromegaly is closely related to myxœdema and cretinism, which have been described in the previous chapter, the diagnosis from these

may be difficult, but in acromegaly the myxœdematous swelling, the coldness and dryness of the skin, the loss of hair, etc., are absent, whilst on the other hand the enlargement and projection of the bony parts does not occur in myxœdema. These morbid conditions are undoubtedly nearly allied.

As regards the relation of acromegaly to giantism, about a fifth of the cases of acromegaly may be regarded as giants. Sternberg also states that many of the giants described in literature suffer from acromegaly. It has been assumed that these conditions are identical, and that giantism is the acromegaly of youth (Klebs, Massalongo, Brissaud and Meige, Hutchinson). Some observations indicate that hypertrophy of the hypophysis may occur in giantism, but this condition also appears under other circumstances, is associated with various disturbances of the general nutrition, and merely creates a disposition for acromegaly (Sternberg).

The close relations between the two conditions have recently been maintained by H. Meige (*Arch. gén. de Méd.*, 1902), Brissaud-Meige (*Nouv. Icon.*, xvii.), Feindel, and Launois-Roy (*Nouv. Icon.*, xv. and xvi.; see also Roy, *Thèse de Paris*, 1903), Medea, Dufraul (*R. n.*, 1904). Launois-Roy were able to demonstrate the persistence of the epiphyseal cartilage by *radiography*, and Cunningham anatomically. But this question is by no means definitely settled, and the relations of giantism to infantilism, as well as the part played by inhibition of sexual development in these conditions, are still unexplained. See also Launois-Roy ("Étude biol. sur les Géants," Paris, 1904.)

A combination of giantism with premature development of the genital system in a six-year-old boy is described by Hudovernig and Popovits (*Nouv. Icon.*, xvi.; see also xix.).

The combination of *giantism* and *infantilism* was present in a case observed by Launois-Roy (*R. n.*, 1902). The extremities in such cases show increased growth in length, and the length of the part of the body below the upper margin of the symphyses is very much greater than that of the part above it. Ossification of the epiphyseal cartilage does not occur, for which reason the growth in length takes place much later than normally. Redlich (*W. kl. R.*, 1906) has contributed a paper upon the subject.

Partial giantism—partial macrosmia—*i.e.* hypertrophy of certain parts of the body (macrocheiria, macropodia, hemihypertrophy, etc.), is distinguished by its congenital development, its limitation to one part of the body, and the excessive deformity of the affected limb. Unilateral giantism has also been described. Hutchinson includes general and partial giantism under the heading of acromegaly, but only some of the cases belong to this class.

Quillou deals with this subject in a thesis. See also Zondek (*A. f. kl. Chir.*, Bd. lxxiv.) and Brüning (*M. m. W.*, 1904).

Circumscribed lipomatosis and *elephantiasis*, or a combination of these conditions, may also give rise to a kind of partial giantism, which is then acquired. Schlesinger describes a peculiar case of partial macrosomia with bulbar symptoms. As regards its occurrence in syringomyelia, see the chapter on that subject.

Osteitis deformans (Paget) is a disease allied to osteomalacia. Its chief symptom is marked distortion of the extremities, especially of the legs, while the hands and feet are unaffected. It also causes considerable increase in the size of the skull, but the changes in the soft parts and the other symptoms of acromegaly are absent. X-ray descriptions have been published by Levi-Londe[1] and Hudelo-Heitz.[2] In these cases

[1] *Nouv. Icon.*, 1897.

[2] *Nouv. Icon.*, 1901.

cerebral symptoms, caused by compression of the cranial nerves, are also present. The disease almost always appears after the age of fifty.

Diffuse hyperostosis is a disease of youth, characterised by marked increase in the size of all the cranial bones. The narrowing of the cranial fossæ, the orbits and foramina, may give rise to blindness, deafness, exophthalmus, headache, dementia, etc. This condition, to which little attention has been given, is extremely rare. Bockenheimer (*A. f. kl. Chir.*, Bd. lxxxv.) has lately devoted a special study to it.

Leontiasis ossea is a tumour-like hyperostosis of the skull, characterised by the formation of bony tumours. But, as Starr remarks, the name is applied to very different conditions. He speaks of a case in which both the cranial bones and the soft parts were affected, as one of *megalocephalia*.

True elephantiasis, pachydermia resulting from local asphyxia, and so-called adiposis dolorosa (see p. 1365), are difficult to diagnose from acromegaly.

Syringomyelia may give rise to enlargement of the distal parts (especially the hands), but this is combined with the deformities and cardinal symptoms of the disease.

Finally, Marie has distinguished a *hypertrophic osteoarthropathy* (secondary hypertrophic ostitis, according to Arnold, with whom Massalongo and Teleky [1] agree) from acromegaly, and has given the following differential signs :—

In osteopathy the end phalanges of the fingers stand out, swollen like drum-sticks, from the large paw-like hands ; the nails are thick, bent (like the beak of a parrot), brittle, and cracked. In acromegaly the swelling affects chiefly the soft parts, in osteopathy mainly the bones. In acromegaly the carpo-metacarpal joint is swollen and thickened, and in osteopathy it is generally unaffected. The lower jaw is always involved in acromegaly, in which there is also thickening of the spine and cervico-dorsal cyphosis, whilst in osteopathy the lower jaw is normal and the cyphosis, if present at all, affects the lower segments of the spinal column. Examination with X-rays reveals differences between these two affections, as Thayer,[2] Schittenhelm, Raynaud-Audibert, Schlagenhaufer,[3] and others have shown.

Osteoarthropathy usually develops in patients suffering from lung diseases (bronchiectasis, empyema, tuberculosis, sarcoma, etc.), and according to Marie it is the toxic bodies circulating in the blood which produce these changes. Further experience has shown that other processes, such as heart-diseases, chronic nephritis, cholæmia, ulcerating tumours, dysentery, etc., may give rise to the affection. It has been assumed to be due to the liver in a few cases, *e.g.* by Gilbert-Fournier, Taylor, Smith, Klippel, and Parmentier-Castaigne. Lemercier distinguishes between a pulmonary, cardiac, biliary, and diathetic origin. Observations by Hirschfeld, Möbius, and Berent seem to indicate that similar conditions may arise from neuritis. Hirschfeld speaks of a vaso-motor dermatohypertrophy, which leads to enlargement of the hands and feet.

Marie's description of osteoarthropathy has undergone some modifications from later observations. The soft parts have been found to be considerably thickened (Arnold, Teleky, Schittenhelm). Indeed,

[1] *W. kl. W.*, 1897. [2] *Journ. Nerv. and Ment. Dis.*, xxv. ; *Phil. Med. Journ.*, 1898.
[3] *Z. f. Heilk.*, 1904.

in the drum-stick fingers, which are regarded as a mild form of osteo-arthropathy (Bamberger), the bony swellings have been sometimes absent (Dennig,[1] Litten), or there has been rarefaction of the end phalanges (Stöltzing). A congenital form also seems to occur (Oppenheim, Decloux, Lippmann, Lemercier [2]), and possibly an acroneurosis of this nature (Oppenheim). I have also observed the symptom in a case of congenital cyanosis.

Future observations will show whether acromegaly can be sharply distinguished from osteopathy. In any case there are forms of this disease which differ from Marie's type (the affection has been called Marie's disease), and which we cannot at present classify (Daireoff, Hirtz, Gasne, and others).

As regards so-called *dysostose cleido-cranienne*, to which we have referred at another place, see also Villaret-Francoz, *Nouv. Icon.*, xvii.; Voisin, *ibid.*

We have no certain knowledge as to the primary *site* of acromegaly. There are many indications that an affection of the hypophysis is the primary cause of the symptoms. Experimental observations by Vassale and Sacchi [3] seem to show that the hypophysis is a necessary organ, which supplies a specific product to the blood. This view is also held by Caselli [4] and Collin.[5] Pirrone, on the ground of his own experiments, thinks it has an antitoxic function. Cyon regards it as an accessory organ of the thyroid gland: he found in it a chemical body which had an exciting effect upon the branches of the vagus to the heart.

In a recent paper he thus defines his standpoint: All increase of the cranial pressure produces *mechanical* stimulation of the hypophysis, which results in a stronger and slower beat of the heart; the rapidity of the venous circulation is thereby increased, especially in the veins of the thyroid, and the brain is thus relieved from the abnormal repletion of blood. The hypophysis in addition produces substances which excite the vagus and accelerator centres, and thus increase the rate of the venous blood-supply. Cyon has also seen erections, polyuria, etc., in stimulation of the hypophysis. In acromegaly we are dealing, not with an increase or decrease of the function of the hypophysis, but with a fully developed disturbance of the function of this organ. See also Cyon, "Les Fonctions de l'Hypophyse," *Acad. des Sc.*, 1907; Thaon, abs. *R. n.*, 1907, etc.

Friedmann and Maas,[6] on the other hand, say they have removed this organ from animals without producing any severe symptoms. Lomonaco and Rymberk [7] have had similar results. They conclude from their experiments that the hypophysis is a rudimentary organ of no functional importance, its excision producing neither symptoms due to injury of the neighbourhood, nor those caused by shock or infection. Rossi and Corning have come to the same conclusions by means of morphological analysis. Friedmann has also found that young, growing cats can dispense with the hypophysis.

There is no doubt that tumour-like degeneration of this gland may exist without acromegaly developing. Of course the tumour in that case is as a rule a destructive one, whilst the tumours which produce acromegaly are usually associated with increase of the glandular tissue

[1] *M. m. W.*, 1901.
[2] *Thèse de Paris*, 1902.
[3] *Arch. ital. de Biol.*, 1893; *Riv. sper.*, 1894.
[4] "Stud. anat., etc., della gland. pit.," 1900.
[5] *Arch. ital. di biol.*, 1899.
[6] *B. k. W.*, 1900 and 1902.
[7] *Riv. mens. di neuropat.*, 1901.

(Timburini,[1] Hutchinson, Schupfer, Hanau, Gubler,[2] etc.). Benda, in particular, maintains, on the ground of his investigations, that acromegaly is caused by an increase of the specific glandular elements of the hypophysis—by a *pathological exaggeration of its function*, whilst others regard absence of the hypophyseal function as its cause. I have seen two cases in which hydrocephalus, due to a cerebellar tumour, had caused the floor of the third ventricle to exercise pressure upon the hypophysis, and thus produced symptoms of acromegaly. A few writers, such as Arnold, Strümpell, Mitchell, and Le Count, regard the enlargement of the hypophysis as merely an associated symptom ; Vassale thinks it a secondary symptom, and Thom expresses himself with great reserve as to the relations. The part played by the thyroids in this disease is also obscure. Many observations point to definite relations between this gland and the hypophysis, which Virchow had previously suspected, and which Vassale, Sacchi, and Rogowitsch confirmed by experiment. It is specially worthy of note that atrophy of the thyroids may cause secondary hypertrophy of the hypophysis (Stieda,[3] Eiselsberg, Hofmeister). The theory was therefore advanced that disturbance in the function of the hypophysis, thyroid, and thymus (possibly also of the sexual glands) might play a more or less important part in the origin of acromegaly (Pineles,[4] Mendel, Parhon-Goldstein, Lorand).

Treatment.—Treatment is still in the experimental stage. Thyroid and pituitary extracts have been given internally, without any evident or constant result (Putnam, Bramwell, Ransom, and others). Cyon, Lancereaux, Faworski, Warda, and Castiglioni report benefit from the use of pituitary extract. I have also seen improvement from this drug in a few cases. Cross [5] found treatment with thyroid and pituitary extract to have a very marked and favourable effect.

Caton and Paul [6] have tried direct excision of the pituitary tumour. The patient died three months later. Others (*e.g.* Eulenberg) have employed surgical treatment, and, within the last few years, with some temporary effect. I would specially refer to the papers by Horsley, Schloffer,[7] Eiselsberg and Frankl-Hochwart,[8] and Hochenegg.[9]

Iodide and mercury are said to have had a good effect in some cases ; I have found the former beneficial. Phosphorus and oxygen have also been recommended.

[1] *C. f. N.*, 1894.
[2] *Korresp. f. Schweiz.*, 1900.
[3] *Zieglers Beitr.*, 1890.
[4] *Volkmanns Samml.*, 1899, N. 242. See also Narbut, *Inaug.-Dissert.*, St Petersburg, 1903.
[5] *Br.*, 1902.
[6] *Brit. Med. Journ.*, 1893.
[7] *Beitr. z. kl. Chir.*, Bd. L., and *W. kl. R.*, 1907.
[8] *N. C.*, 1907 ; *W. m. W.*, 1907 ; *Z. f. N.*, xxxiv. For an account of the methods, see Loewe (*N. C.*, 1907).
[9] *D. m. W.*, 1908.

CONDITIONS OF INTOXICATION

WHICH SPECIALLY INVOLVE THE NERVOUS SYSTEM

Alcoholism

Acute alcoholic intoxication need not be described here.

The chronic abuse of alcohol has a very marked influence upon the central nervous organs and upon the peripheral nervous system. It affects the *functions of the brain* in various ways. The nature and character undergo a radical change, which becomes specially apparent as regards the *moral* sense. Excitability, emotional weakness, quarrelsomeness, a tendency to outbursts of rage and violence, indolence, neglect of duties, indifference to family affections, offences against propriety and decency, are the usual characteristics of an habitual drinker.

Intolerance of alcohol may develop, so that small quantities may have a toxic effect and produce pathological intoxication. We cannot here consider the forensic and criminal importance of alcoholism. Prinzing, Sullivan, and others have, for example, showed by statistics the part it plays in the increase of suicide. Marked *dementia* often appears in the advanced stages, and it may last for years and pass into idiocy. Of the *acute psychoses* which develop in the course of alcoholism, we shall only here discuss the *intoxication-psychosis*, par excellence, viz., delirium.[1]

The symptoms of the various *cerebral disorders* produced by chronic alcoholism correspond in many respects to those of the neuroses—epilepsy, hysteria, and neurasthenia. Epilepsy is often the result of alcoholism. About thirty per cent. of the alcoholics received into the delirium ward of the Charité suffered from epilepsy. It differs from genuine epilepsy only in its late development, and in the fact that it usually disappeared when alcohol was withdrawn. The attacks were specially apt to follow great alcoholic excess. Some writers (Féré, Magnan, Warthmann [2]) regard alcoholism as merely the exciting cause of the epilepsy, but this view does not seem to me to be in accordance with the facts (see also p. 1201). An epileptic attack frequently ushers in delirium tremens. This form, however, is distinguished by Bratz from

[1] Bonhöffer has thoroughly studied these conditions in his works: "Die akuten Geisteskrankheiten der Gewohnheitstrinker," Jena, 1901 (Fischer). They have also been discussed in numerous recent papers (Luther, Wollenberg, Hoppe, Hirschl, Cramer, Heilbronner, Mayet, etc.). Compare also H. Hoppe, "Die Tatsachen über den Alkohol," second edition, Berlin, 1901; E. Meyer, *A. f. P.*, Bd. xxxviii.; Goldstein, *Z. f. P.*, Bd. lxiv. Chotzen, *A. f. P.*, Bd. xli.; and with regard to the forensic questions: Leppmann, "Alkoholismus und Ehescheidung," *Ärztl. Sachverst.*, 1905; Strassmann, *ibid.*; Heilbronner, "Die strafrechtl. Begutachtung der Trinker, Samml. zwangl. Abhandl.," Halle, 1905; Endemann, "Die Entmündigung wegen Trunksucht," etc., Halle, 1904; Bonhöffer, "Die alkohol. Geistesstörungen," *Deutsche Klinik*, 1905; and also the text-books on Psychiatry by Krafft-Ebing, Kraepelin, Ziehen, Binswanger-Siemerling and others.

[2] *A. f. P.*, xxix.; see also Neumann, "Beziehungen zwischen Alkoh. und Epilepsie," 1897.

the true, grave form, which occurs after years of alcoholic abuse, on account of its more favourable prognosis.

The *subjective symptoms* of alcoholism are in many respects similar to those of hysteria and neurasthenia. The patient complains frequently of anxiety, depression, sleeplessness, restlessness, tremor, general weakness, pain, loss of appetite, palpitation of the heart, etc. The *objective* symptoms may, as in neurasthenia and hysteria, include *exaggeration of the deep reflexes, of the mechanical excitability of the nerves and muscles, hyperidrosis, hypæsthesia*, and *anæsthesia* of the sensory type, *vasomotor disorders*, etc. The *convulsive attacks* may greatly resemble those of hysteria. *Unilateral convulsions* and *paralyses* also occur; these may regress and, so far as our experience goes, they are not due to any recognisable pathological changes. Status hemiepilepticus frequently develops on the same basis. Tetanoid spasms without loss of consciousness, and resembling those of strychnine poisoning, have been described by Siemerling. Tetany may also be due to alcoholism (Brandenburg).

Alcoholic tremor, one of the most common symptoms, is usually more intense and coarse than the tremor of the neuroses; it affects not only the extended fingers, but specially involves the lips and tongue, and tends to be most marked in the morning before the patient has taken food. It accompanies voluntary movements, although it is not strictly combined with them.

Quinquaud has described another sign: If the physician places the spread-out fingers of the patient perpendicularly upon the palm of his hand, he will feel a slight quivering, as if the fingers jerked against each other—a kind of jarring or crepitation. Quinquaud's sign, which has been studied by Maridort, Aubry, Fürbringer (*B. k. W.*, 1905), and Hoffmann-Marx (*B. k. W.*, 1905), is said to be most distinct in grave alcoholism, but the investigations of Lauscher (*B. k. W.*, 1906) and Minor (*B. k. W.*, 1907) show that it also occurs under other conditions, and should not be regarded as pathognomonic.

The *gastric disorders* may also be of nervous origin, but a true *gastritis* or chronic catarrh of the stomach may be present. *Morning vomiting*—nausea and the vomiting of slimy masses in the morning—and *anorexia* are very characteristic.

Visual disturbances, which are very common, may be of a purely functional nature (concentric narrowing of the field of vision), or may be caused by *optic neuritis* or by *partial atrophy of the optic nerve*, with pallor of the temporal half of the discs, as Uhthoff[1] has specially described. *Central scotoma* for colours (red and green, see p. 515) is the ordinary form of this optic nerve affection.

As regards the condition of the pupils in alcoholism, see the papers by Thomsen, *Charité-Annalen*, xi.; Moeli, *B. k. W.*, 1897; Uhthoff, *B. k. W.*, 1886; *Graefes Arch.*, Bd. xxxii., and *Graefe-Saemisch Handbuch*, xi.; also Bumke, "Die Pupillenstörungen," etc., Jena, 1904; Kutner, *D. m. W.*, 1904; see p. 514.

Other symptoms, which may occur in alcoholism, are the result of *peripheral neuritis* due to the poisoning. Slight degrees of this neuritis are very common; they are probably the cause of the *cutaneous and muscular hyperæsthesia*, the *pain* in the legs, which is often so acute, and the objective sensory disturbances which sometimes appear in the parts supplied by the peripheral nerves, *e.g.* the external cutaneous of the thigh. It is not certain whether the *cramp in the calves* and similar tonic muscular

[1] *Graefes Arch.*, Bd. xxxii. and xxxiii., and *Graefe-Saemisch Handbuch*, xi.

contractions are due to slight inflammatory changes in the nerves and muscles, or not. Under the influence of certain factors, this neuritis, which is to a certain extent latent, may develop into true alcoholic polyneuritis (see chapter on multiple neuritis).

The various conditions produced by alcoholism may combine in numerous ways, and the effect of the poison upon the *cardiac and vascular system*, the *liver*, *kidneys*, etc., may give rise to symptoms which often produce an *exceedingly varied clinical condition*. Smith would attribute the feeling of dread and other uncomfortable sensations in alcoholics to weakness of the heart and atony of the vessels, but this only explains one form of the disturbance.

The severe effects of alcoholism are particularly noted in *spirit drinkers*, but wine and beer drinkers are by no means exempt. Some of the symptoms, such as multiple neuritis, are specially apt to follow excessive beer-drinking.

Alcoholism is naturally most apt to develop in individuals in middle life, between the ages of thirty and fifty, but no age is quite exempt.

Convulsions and other signs of alcoholism have been seen to occur in infants fed by a drunken mother or wet-nurse (Meunier, Combe). Ladrague, Kassowitz (Berlin, 1902), Grósz, and others have published papers upon the alcoholism of childhood.

Individual idiosyncrasy enters largely into the matter of susceptibility to alcohol. In some cases a small amount of alcohol, taken regularly, acts as a poison, whilst in others ten times the quantity can be borne. Trauma lessens the power of the nervous system to resist this poison.

Drunkenness is often a *symptom of the neuropathic disposition*, or of degeneration, and persons so constituted are specially liable to develop alcoholic psychoses. It has been stated elsewhere that alcoholism in the parents constitutes a grave danger for the health of their children, and creates the tendency to neuroses or psychoses or to degenerative conditions of many kinds.[1]

Dipsomania has been classed by Kraepelin, Aschaffenburg, and in particular by Gaupp,[2] who has devoted special study to it, along with epilepsy; according to them it is a form of mental epilepsy. The condition is characterised by attacks of depression, which give rise to the overmastering impulse to consume intoxicating liquors. This leads to great alcoholic excess, accompanied or followed by more or less profound unconsciousness. The attack lasts for days or weeks.

The history of Fritz Reuter and the description which he himself gives of his disease in a letter to his cousin (see Theod. Gaedertz, *Nationalzeitung*, issue of the 13th July 1904), shows that this condition may arise in men of high mental and ethical character. See also the study of Albrecht, "Fritz Reuters Krankheit," Halle, 1907.

Delirium tremens requires special consideration. It may appear at any time in the course of alcoholism, and show frequent *relapses*. Cases

[1] We may refer in this connection to the interesting communications of Bunge upon alcoholism and degeneration, and to the works of Magnan ("De l'alcoolisme," 1874), Bär, Arrivé, Kende, Bourneville, Rabinovitsch, Strohmayer, Bezzola, Garnier, Crotters, Anton, Laitinen (*C. f. N.*, 1906). Kraepelin and his pupils have specially contributed to put the campaign against the abuse of alcohol upon a scientific basis. See also H. Hoppe, "Die Tatsachen über d. Alkohol," second edition, Berlin, 1901; E. Hirt, "Der Einfluss d. Alk. auf das Nerven- und Seelenleben," *Grenzfragen*, etc., 1904; Ziehen, "Über d. Einfl. d. Alk. auf d. Nerv.," second edition, Berlin, 1904; Helenius, "Die Alkoholfrage," Jena, 1903; Horsley-Sturge, "Alcohol and the Human Body," London, 1907.

[2] "Dipsomanie," Jena, 1901.

have been known in which the same person had twenty to twenty-seven attacks of delirium. It has been wrongly assumed that sudden abstinence from alcohol, which has previously been taken regularly, may bring it on. A toxic effect has been thought by some writers, such as Wagner, Kraepelin, Jolly, Herter, Elsholz, to be the cause, and the latter believes alcohol to be the antitoxin of this poison in the body, for which reason abstinence is thought to be capable of bringing on the delirium. The condition rarely occurs without a cause, and is usually brought on by some *exciting factor*, such as repeated, severe alcoholic excess, trauma and febrile diseases (especially pneumonia). The first symptoms are *loss of appetite*, *marked tremor*, motor and mental *unrest*, hurried movements and depression. *Sleep* then becomes affected and is disturbed by horrible dreams; the patient finds great difficulty in going to sleep, as mere closing of his eyelids brings on terrifying visions. *Speech* may, during the whole delirium, be affected by the tremor of the lips, and may show a kind of syllable-stumbling, like that of paralysis.

The delirium is shown by motor and mental restlessness; the tremor becomes greatly increased, and *innumerable hallucinations* and *illusions* take possession of the patient's mind. The sensory hallucinations and fixed ideas are usually related to real impressions. In the pattern of the bedcover he sees lice, spiders, etc., which are running about; the figures in the carpets give him similar ideas. At first, he is conscious of his delusions every time he opens his eyes, but before long he becomes completely dominated by the hallucinations. He is quite *confused* and *incoherent*, and the fancies called up by the sensory hallucinations follow thick upon each other. In addition there is great *motor restlessness*, shown by the way in which he throws his hands about, pulls at the bedclothes, and seizes any object within his reach. He is constantly driving away or catching *animals*, trying to pull threads, worms, etc., from his mouth, or to protect himself from persons who are seeking to kill him or give him poison. Bonhöffer [1] regards the combined appearance of hallucinations relating to various sensory regions as peculiarly characteristic. The content of the imaginations changes rapidly, but almost always has a *comical*, *grotesque*, and *terrifying* character. The anxiety and fear which dominate the patient are often contrasted with the marked hilarity which he exhibits. He either cannot be snatched out of his delirium, or forgets it only for some moments, during which he is docile and gentle. He is seldom aggressive.

During the delirium the appetite is lost, the pulse is rapid, reaching a hundred and twenty to a hundred and fifty beats, and is sometimes dicrotic; the *temperature* is usually raised—in 80 to 90 per cent., according to Döllken—but this increase is often a result of the accompanying affection of the respiratory or digestive systems, or infection from some wound (Bonhöffer, Ziehen). On the other hand some severe forms of delirium are from the first associated with very high fever; this is the febrile alcoholic delirium described by Magnan and Laségue and mentioned by Alzheimer.[2] Elsholz found increase of the leucocytes and excess of polynuclear neutrophile cells over the mononuclear in the blood at the height of the delirium. He has also mentioned a form of conjunctivitis as a symptom of delirium.

[1] "Der Geisteszustand der Alkoholdeliranten," 1897, and *M. f. P.*, i.
[2] *C. f. N.*, 1904.

The patient is insensitive to pain, and moves the injured limbs about. For this reason *pneumonia* is often overlooked, as he neither complains of pain nor breathes superficially. Laitinen has shown experimentally that the use of alcohol increases the disposition to infective diseases.

The patient usually *perspires* heavily. The urine is scanty, and in almost fifty per cent. of the cases contains a slight trace of *albumen*. According to Hertz, albuminuria is an almost constant symptom; he assumes that there is a genetic connection between the kidney affection and the delirium. Döllken [1] also finds albuminuria as a constant symptom. Albumosuria is less common (Liepmann). In many cases there is involuntary loss of urine and fæces, but no retention of urine.

The delirium reaches its height in two to three days. As a rule it lasts for three to six days, and usually ends in a *deep sleep of six to twelve hours' duration*, from which the patient wakens strengthened and almost clear in mind. The pulse again becomes full and slow, and the temperature normal. Tremor and restlessness may persist for a short time, and there is a dream-like, partial recollection of the delirium. In less favourable cases this condition continues until a fresh sleep brings recovery.

Finally, there are some cases in which the delirium persists and the patient dies from *collapse*. The pulse becomes smaller and more rapid, the temperature remains high or suddenly rises to a great height. The prognosis is specially gloomy if *cardiac weakness*, pneumonia, nephritis, trauma, etc., are also present.

According to the available statistics, death occurs in about 15 per cent. of the cases of delirium tremens. Jacobson (*Z. f. P.*, Bd. liv.) puts it at 13 per cent., Bonhöffer at 9 per cent. (or 1 per cent. in prison), whilst Villers calculates it at only 1·5 per cent. in Brussels, from which he concludes that the affection assumes a mild form in that town. The percentage of deaths from delirium tremens is also very low in Wagner's clinic (Pilcz), and, according to Eichelberg's latest statistics, in the Eppendorfer Hospital (*M. m. W.*, 1907).

It is an unfavourable sign when the pulse-rate and temperature do not diminish after the critical sleep. A condition of *hallucinatory confusion*, of *insanity with delusions of jealousy*, or dementia may follow the delirium. Kraepelin speaks of the weak-minded terminal conditions of delirium, and E. Meyer describes a *paranoid dementia*. Krukenberg has shown that concentric narrowing of the field of vision is often present during, and eight to fourteen days after the delirium. It would also seem that localised cortical cerebral symptoms may in rare cases develop at the height of the delirium (Bonhöffer). Hasche-Klünder [2] reports cases which show that alcoholic delirium may simulate organic brain disease.

Cramer gives the following diagnostic symptoms of *pathological intoxication*: conditions of anxiety and delirium, sensory hallucinations, sluggish reaction of the pupils, tendency to acts of violence, all terminating in sudden collapse and sleep, complete or partial amnesia, etc. In most cases there is evidence of a degenerated constitution.

We cannot here discuss Wernicke's acute hallucination, chronic delirium, or Korsakow's psychosis (see p. 514).

The *pathological changes* found in the central nervous system in

[1] "Die körperl. Erscheinungen des Del. trem.," Leipzig, 1903.
[2] *Mitt. aus d. Hamb. Staatskrank.*, 1905.

alcoholism are on the whole slight; they consist merely of hyperæmia of the cerebral membranes, slight cloudiness, occasional œdema of the arachnoid and pia, and external hydrocephalus. *Hœmorrhagic pachymeningitis* is not uncommon. The brain is usually normal, but sufficient investigation has not yet been made as to the fine histological changes. Berkley has found changes in the nerve-cells by recent methods, and Bonhöffer has reported similar changes, in addition to disintegration of the myelin, in the radial fibres of the motor cortex and cerebellum, especially of the superior vermiform process, shown by the Marchi method; the value and significance of these changes are, however, very doubtful, as Bonhöffer himself recognises, at least as regards the cell changes shown by the Nissl method. He also points out that in severe cases of alcoholic delirium the central grey matter is the favourite site of hæmorrhagic infiltration. See also Robertson (*Brit. Journ. Inebriety*, 1904).

Treatment.—The most important point is *prophylaxis*. Much has been done during the last twenty years in this respect, but much still remains to be done. A number of physicians, *e.g.* Forel, Kraepelin, Bunge, Baer, Delbrück, and others, have, partly by their personal influence, partly by their scientific work, placed the struggle against the abuse of alcohol upon a proper basis, and have endeavoured to spread the knowledge of its injurious effects. The various total abstinence and temperance societies (Blue Ribbon Association, the order of Good Templars, etc.), and the association for the prevention of the abuse of spirituous liquors have been of special use in this respect. The Imperial Board of Health has, on the recommendation of these Societies, issued a leaflet on the subject.

The occurrence and spread of alcoholism will be to a certain extent restricted by the public-house reforms, the founding of temperance hotels, reading-rooms for the people, inquiry and refuge offices, improvement of the housing of the poorer classes, etc., and not least by abolition of the custom of drinking.

See the interesting statements by Laquer, *Ther. d. Geg.*, 1908.

The proper *treatment* of alcoholism is *withdrawal of the alcohol.* This is most difficult to carry out when the disease arises out of primary degeneration. The drunkard can only in very rare cases attain this object by his own strength of will. The withdrawal must, as a rule, be carried out in a *hospital.* Certain parts of hospitals or asylums are set aside for this purpose, but it is a more general and growing custom to send such cases to special *institutions for inebriates.*

The principle of the modern *institution for inebriates* is complete and total abstinence of the patient and those around him, and his education to maintain this abstinence throughout his whole lifetime. We might name the institutions of Lintorf in Düsseldorf, Ellikon in Zürich, Waldesruh in Hamburg, Waldfrieden in Fürstenwalde, and the private homes of Fürer in Rockenau, Schmitz in Bonn, Colla in Buchheide, Römer in Elsterberg, Smith in Niendorf, Clemenz in Schönwalde, etc.

The reports of various physicians and directors, comprising the result of much experience, are unanimous in stating (1) that the cure by withdrawal of alcohol requires a considerable time—on an average from six to nine months to a year, and (2) that permanent results can only be attained by *complete abstinence*, which must be continued after the patient's discharge for the whole of his lifetime (Forel, Kraepelin, Bresler, Bregmann,

Moeli, Nonne). The societies already mentioned, especially the order of Good Templars, are extremely helpful in this respect. In 1903, it had in Germany 637 lodges, and 22,355 members.

There are still great defects in the regulation by law of *care of inebriates*. The fact that a patient cannot be forced into an institution for treatment without being certified is a factor very much against his recovery.[1] The necessity for a law as to the care of inebriates upon another basis and for the founding of state institutions where the inebriate can be confined is becoming more and more urgently felt. The Berlin Psychiatric Society, Endemann, Colla, Delbrück, Nonne, and others have emphasised this necessity. The latter has published valuable rules for the organisation, management, and conduction of institutions of this kind (*Handbuch der sozialen Medizin*, Bd. iv., Teil 2).

Further experience of treatment by *hypnotism* is required to determine its value. Forel, Ouspenski, and others say they have effected a cure by means of suggestion.

An attempt has been made to excite an antipathy for alcohol by adding strychnine or antimonial wine to the intoxicating liquors. The so-called *gold-cure*, comprising internal administration of chloride of gold and other drugs (Fenn, *Brit. Med. Journ.*, 1904), and the subcutaneous injection of atropin (along with cinchona given internally), etc., are of no practical value.

The withdrawal of alcohol often cures the severe mental disturbances, such as the dementia, but I have seen a few cases in which abstinence and treatment only influenced the physical symptoms, *e.g.* the polyneuritis, whilst the mental impairment persisted.

The delirium calls for absolute and immediate abstinence. Alcohol should only be given if pneumonia sets in or a collapse is threatened; in such cases it is often impossible to withhold alcohol. Förel would not make even this exception. A strengthening diet is necessary. Stimulants have also been recommended, (Quénu uses injections of sperminin, which Massenier has also tried.) Ganser[2] has found *digitalis* beneficial.

Delirious patients should be treated in an institution where *isolation* can be carried out with all the necessary precautions, and where it is not necessary to restrain the patient mechanically.

The *bromides* are useful in slight delirium. Narcotics should be used with great caution. *Chloral hydrate* is preferable to opiates, but it should only be given at long intervals and with careful watching of the heart. Wagner and Pilcz do not use this drug, but they think it very important to give calomel as a purgative.

Strychnine has been recommended for the alcoholic tremor. I have found that the bromides often have a favourable effect upon this tremor.

[1] Para. 6, section 3, of the German Civil Code runs as follows: "A person can be interdicted if, on account of habitual drunkenness, he is unable to manage his affairs, or if he exposes himself or his family to the danger of want, or endangers the safety of others."

[2] *M. m. W.*, 1907.

Morphinomania

Literature: Levinstein, "Die Morphiumsucht," third edition, Berlin, 1883; Erlenmeyer, "Die Morphiumsucht und ihre Behandlung," third edition, 1887; Crothers, *Quarterly Journ. of Inebriety*, 1892; Obersteiner, *W. kl. W.*, 1888; Dizard, "Étude sur le morphinisme," etc., 1897; Rodet, "Morphinomanie et morphinisme," 1897; Jastrowitz, "Über Morphinismus," *Deutsche Klinik*, etc., vi.; Pouchet, "Morphinomanie," etc., *Prog. méd.*, 1898; Crothers, *Med. Rec.*, 1899; Mills, *Internat. Clinics*, 1905; Brouardel, "Opium, morphine, et cocaine," etc., Paris, 1905.

Morphinomania is the name which Levinstein has given to the *craving* of an individual to use morphia as a means of stimulation or gratification to such an extent that abstinence from it gives rise to subjective and objective derangement of the general health, and also to the *morbid condition* which is produced by the improper use of the drug.

This condition is practically always caused by *subcutaneous injections of morphia*, very rarely by its internal administration.

The development of the condition is usually as follows: The morphia is originally employed to control some physical or mental pain. From its continuous and regular use the body becomes accustomed to the poison,[1] pleasure in it gives way to dependence upon it, and as the original dose no longer suffices to bring on a condition of euphoria it is gradually further and further increased. In the end the patient becomes incapable of any mental activity, of attending to his business or meeting other people unless he has previously taken the quantity of morphia which gives him the requisite mental and physical energy. This craving for morphia is accompanied by many other general disturbances (tremor, gastric symptoms, coughing, etc.), which only disappear after a fresh injection of the poison.

The cases in which morphia is taken regularly for weeks or months on account of acutely painful conditions, and is abandoned as soon as the pain has ceased or the symptoms become transient, should not be regarded as morphinomania, even although signs of chronic morphinism may have appeared.

Physicians are the most frequent victims of this disease. Among two hundred and fifty morphinomaniacs, ninety-three were of the medical profession (including women-doctors). Of a hundred male patients, forty-two were physicians. *Pharmacists* are next most liable to the disease. On the whole, I am inclined to think that the trouble has become less common during the last twenty years.

Many of those afflicted do not suffer from any chronic, incurable disease associated with great pain; it is rather those troubled with neurasthenia, hypochondria, depression, and so on, who have themselves recourse to morphia or persuade their physician to give it to them.

Naturally morphinomania hardly ever develops unless the drug produces a *condition of euphoria*, or a kind of *sensory intoxication* which is associated with a feeling of great contentment. As soon as this intoxication wears off, the usual depression or pain, which has been banished, again returns, and the longing or craving for morphine again awakens. And

[1] It is doubtful whether this habituation is due to the formation of protective bodies (antitoxins) in the body, as Hirschlaff and Gioffredi conclude from their experimental investigations (Morgenroth). Faust assumes that the poison is destroyed in the body by oxidation. See also Cloetta, *A. f. exp. Path.*, Bd. l.

as the individual affected is originally weak-willed, or has become so on account of his long morphia-habit, he becomes more and more a slave to it. It is easy to understand why the physician, who always has the drug at hand, and who is compelled by his profession to be continually at his post, should fall an easy victim to this habit.

The average *dose* injected is about 1·0 g. (15 grains) per day, but cases have been known in which 3·0 to 5·0 g. (45 to 75 grains) have been injected daily. In some cases other drugs are habitually used (cocaine, chloral hydrate, chloroform, ether, etc.).

Sooner or later, sometimes after the drug has been used for six to eight months, sometimes for a year or more, the *symptoms of chronic morphia poisoning* appear. The *nutrition* suffers, the fatty tissue gradually disappears, the skin becomes flaccid and loose, the face pale, ashen-grey, or sometimes dark red. The *pupils* are usually narrow and react sluggishly to light. Diplopia and *paresis of the accommodation* sometimes occur. *Hoarseness, thirst, loss of appetite, tremor and disturbances* of speech are common symptoms. *Depression, restlessness*, and inability to do mental work also develop, but they may be held for a long time at bay by the morphia injections. Each injection leaves the patient rejuvenated and able to meet the utmost demands upon his mental and physical powers.

The *demoralising* effect of the morphia habit is well known. The patient becomes untrustworthy, untrue to himself and to others; his moral sense becomes duller and duller. A true intoxication-psychosis develops in rare cases. Transient conditions of *drowsiness*, slight confusion with sensory hallucinations or *attacks of syncope* may occur.

As a rule there is obstinate *constipation*, and *gastric disorders* may develop. *Impotence* and *amenorrhœa* are constant symptoms. *Azoospermia* has been observed. The secretion of urine is usually diminished, and *albuminuria* has been repeatedly found.

Febrile attacks, an intermittent type of fever with a rise of temperature to 38·5 or 40·0° C. (102 to 104° F.), and enlargement of the spleen, etc., have occasionally been observed.

If the morphia habit becomes still more firmly rooted, a condition of *marasmus* ultimately develops, from which the patient dies. Multiple *abscesses* are often produced by the injections, and have an unfavourable effect upon the general condition. Infective diseases, particularly pneumonia, are apt to prove fatal in morphinomaniacs.

The symptoms which follow withdrawal of the morphia are so typical that they are known as the *symptoms of withdrawal*. Within a few hours after the last injection, the patient becomes *restless, anxious*, and *excited*, and the craving for morphia awakens. He cannot sleep, jumps out of bed, and shows an *imperative impulse for movement*. *Nausea, sickness, vomiting, incontrollable restlessness, diarrhœa, palpitation of the heart*, and a *rush of blood* to the head, soon appear. Among the later symptoms are *hyperidrosis, salivation, coryza*, yawning, coughing, tremor, and neuralgic pains in various parts of the body. *Paresis of the accommodation* is almost constantly present; the pupils are dilated and often unequal and springing. The restlessness and tremor increase, the speech becomes more difficult (resembling that in paralysis), and finally lalling. Conditions of hallucinatory excitement and attacks of mania are not uncommon during the period of withdrawal. Epileptiform attacks and choreic conditions are

unusual. In a case of Hochstetter's[1] (Jolly), a relapse of the chorea coincided with the period of abstinence. I have in one case seen marked motor ataxia develop in the limbs when the morphia and heroin were withdrawn; this disappeared after an injection of morphia.

Levinstein describes delirium which closely resembles that of alcoholism (Abraham[2] gives some of the differentiating signs). This may be followed by a stage in which the patient is under the control of some fixed delusion, especially of a hypochondriacal character, but it only lasts for a few days.

Chotzen (*Z. f. P.*, Bd. lxiii.) discusses the mental disturbances which occasionally occur in abstinence from morphia.

With regard to chloral delirium, see Antheaume-Parrot (*L'Encéphale*, 1906).

Collapse is one of the most dangerous symptoms of withdrawal. The pulse becomes suddenly irregular and slow, falls to forty, or even thirty beats; the patient becomes unconscious and breathes with difficulty; his face grows pale and sunken, etc. If this condition is not immediately treated, death takes place. The collapse occurs about five days after the morphia is stopped. There may be repeated attacks of slight or severe syncope. A condition allied to dipsomania (see previous chapter), in which the patient only resorts to morphia for a time on account of periods of depression, has been described by Krafft-Ebing as morphinodipsia.

In a case of Manchot's there was a marked tendency to cutaneous hæmorrhages during withdrawal (urticaria factitia hæmorrhagica).

Sexual desire is usually markedly increased during the first days of withdrawal.

The *prognosis* is unfavourable. Although the morphia can usually be withdrawn, there is great danger of a relapse. Among eighty-two of Levinstein's male patients, sixty-one relapsed; among twenty-eight female patients, ten; and among thirty-two physicians, twenty-six were affected a second time. The prognosis is therefore particularly unfavourable in the case of medical men. The prospects of complete recovery are most hopeful in patients who have used morphia on account of some disease which has been cured. Hirt reports twenty-seven cures in thirty-five cases, a percentage, therefore, of seventy-seven.

Treatment.—Prophylaxis is a matter of great importance. One cannot, in this respect, too urgently protest against the inconsiderate use of morphia and opiates. The physician should, above all, refuse to allow the syringe to pass out of his own hands, unless in cases which are bound before long to prove fatal. If he is obliged to give subcutaneous injections for any length of time on account of some painful condition, he should withdraw the drug as soon as he possibly can. The greater the euphoria produced by morphia, the greater is the care required in its use.

The only treatment of morphinomania is that of *withdrawing* the drug. Unfortunately this has only in a few cases led to definite cure. Obersteiner is, however, wrong in limiting the indications so very narrowly as he does. Levinstein approves of *sudden withdrawal*, and only resorts to the *modified* course of treatment under certain conditions, *e.g.* if the morphia habit is associated with severe disease of the internal organs and a collapse may be feared. In the modified method, the full dose is at first given, and then a certain portion of it is given for a few days. Other

[1] "Inaug.-Diss.," Berlin, 1894.

[2] *C. f. N.*, 1902.

physicians, who have experience in this matter (Burkardt, Müller, Obersteiner, Pressey,[1] etc.), approve of *gradual* withdrawal.

The morphia can hardly be cut off except in some *institution*, where the patient can be most carefully watched during the whole period. Hirt[2] does not agree with this principle; he would, at least, carry out the withdrawal outside an institution, in patients who inject less than 4 to 6 grs. per day. I have met in my practice with five or six cases in which patients of very strong character have permanently cured themselves of the morphia-habit, and Deutsch[3] has also seen such cases.

The *suicidal impulse* is not infrequently aroused, and very special supervision is necessary in such cases.

Whilst the morphia is being withdrawn, great care must be taken to maintain the strength, and for this reason it is often impossible to avoid giving *alcohol* (champagne, port-wine, brandy, etc.), but these should be prescribed only when they are absolutely necessary. Various drugs, such as cocaine, spartein, nitro-glycerine, etc., have been recommended as substitutes, but cocaine should be absolutely avoided. It certainly facilitates the withdrawal of the morphia, but it gives rise in itself to severe mental symptoms—*cocaine-psychosis*—which are characterised as a special form of hallucinatory paranoia. If cocaine is given, the morphinomaniac often becomes a victim to the cocaine habit.

Chloral-hydrate and paraldehyde may cause delirium. Codeine, belladonna, bromides, quinine, etc., may be tried, but should be used only for a short time. Pelz has described a condition of *codeinism*.[4] Fromme recommends *dionin* as a substitute; he gives it in doses of $\frac{3}{4}$ to 1 gr., and injects as much as 15 grs. per day. Heroin has also been used in this way, but I have seen unpleasant after-effects from it. Sollier[5] also disapproves of heroin, and describes the danger of heroinomania, which I have also seen and treated several times. So-called antimorphin is rejected by Levin and others. Hyoscin has also been tried as a substitute, but is not now approved of. The anti-serum with which experiments were made (Hirschlaff), very soon passed out of notice.

Deutsch would replace subcutaneous injection by administration per anum, before the actual withdrawal is commenced. I have also occasionally found withdrawal aided by this method.

Baths with cold douches are beneficial. *General massage* and perhaps hypnotism may be tried for the insomnia, which is often very persistent. Hirt and Bérillon lay stress upon the effect of suggestion, and I regard psychotherapy as a very essential accessory of treatment by withdrawal.

Grave conditions of collapse are most effectively treated by *morphia*. As a rule a comparatively small dose is required ($\frac{1}{2}$ gr.), which should be repeated if it has not the desired effect.

Hofmann (*Therap. Mon.*, 1902) and Erlenmeyer (*Therap. Mon.*, 1903) recommend camphor.

After the morphia has been withdrawn, the patient should still be watched by his physician, and he should not at once return to his ordinary environment and business. It is particularly adyisable to send him for a *sea-voyage*, the doctor on board the ship being carefully instructed about the case. I have succeeded in this way in obtaining a

[1] *Journ. Amer. Med. Assoc.*,,1905. See also Erlenmeyer, *loc. cit.*; Joffroy, *Prog. méd.*, 1899.
[2] *N. C.*, 1898. [3] *W. m. W.*, 1899.
[4] *D. m. W.*, 1905. [5] *Presse méd.*, 1905. See also Comar-Burat, *Presse méd.*, 1904.

permanent cure in one case; in another the patient, whom I permitted after a sea-voyage to spend the winter in Heligoland, took to *chloroform*, which was apparently the cause of his death.

Above all, the patient must never after his recovery be allowed to use morphia on any pretext. Withdrawal of the drug has some effect even in cases which relapse, as small doses have again become sufficient.

Chronic lead-intoxication, which would follow here, has already been discussed on p. 517.

APPENDIX

Tetanus

Literature: Rose, "Über den Starrkrampf," Pitha-Billroth's *Handbuch der Chir.*, Erlangen 1870; Rose, "Der Starrkrampf beim Menschen," *Deutsche Chirurgie*, viii., 1897; Leyden-Blumenthal, "Der Tetanus," *Nothnagels Handbuch*, v., 1900; Behring, *Ther. d. Geg.*, 1900; *ibid.*, "Ätiol. u. ätiol. Therap. d. Tetanus," *Beitr. z. experiment. Therapie*, vii., Berlin, 1904; Rozenraad, "Die neueren Ergebnisse in der Behandl. u. Prophy. d. Tetanus," "Inaug.-Diss.," Leipzig, 1902.

Infection is the most important, and probably the only cause of tetanus. The *bacillus* was discovered by Nicolaier, and Kitasato has made pure cultures of it. It is contained in the earth, enters the body through a wound in the skin, or less often through the mucous membranes (pharynx, uterus), where it also produces a local effect. Tetanus is caused, not by the bacillus itself, but by the *products of toxic metabolism* to which it gives rise—the tetanin discovered by Brieger, tetanotoxin, spasmotoxin, and another very poisonous toxalbumin obtained from pure cultures. A laboratory-infection produced in this way was described some years ago.

We cannot here describe the very important observations of Ehrlich upon this subject, and the side-chain theory which he has advanced, nor the views of other writers (Buchner, Knorr, Dönitz, Roux, Blumenthal), some of which contradict, some of which agree with Ehrlich's teaching. Nor can we discuss Behring's allotropic theory.

Wassermann and Takaki have shown that the central nervous system contains substances which neutralise the tetanus toxin, or have an antitoxic effect (confirmed by Marie, Metschnikoff, and others). Milchner proves that the cerebral nervous substance enters into chemical union with the tetanus poison. These facts have been turned to good account in treatment (see below). Meyer and Ransom (*Arch. f. exp. Path.*, 1903) have shown that the tetanus poison is conveyed by the lymph tracts into the motor nerves, and by means of these to the nerve-cells of the spinal cord. On this subject see Sjövall (*N. C.*, 1904), Zupnik (*D. m. W.*, 1905), and others.

The disease arises mostly from wounds which are contaminated by *earth, splinters of wood*, and other *foreign bodies* (especially horse-dung). The spores of the bacillus may retain their virulence for years upon the foreign bodies. The nature of the wound and the way in which it has been inflicted are of no importance. During the last few years injections of gelatine used for the purpose of checking hæmorrhage have in several instances brought on tetanus (Gerulanos, Georgi, Lorenz, etc.). *Puerperal tetanus* is undoubtedly of traumatic origin, and may be conveyed to the mother by the tamponade, forceps, etc. In one case Heyse found the bacillus in the dirt on the floor. *Tetanus neonatorum* is of the same nature; the bacillus finds its entrance through the umbilical wound, or less

frequently through an incision of the prepuce. In some rare cases (Süss) tetanus is said to have arisen from a gastric disease (?).

Some writers speak of an *idiopathic* or *rheumatic tetanus*, which occurs spontaneously or as the effect of chill. There can hardly be any doubt that in such cases the wound has been overlooked or already healed. An *endemic* and *epidemic* occurrence of tetanus has often been reported. Tetanus is certainly more frequent in the tropics than in Europe.

Symptomatology.—The initial symptoms seldom develop in less than five or ten days after the injury, and in some cases several weeks may elapse before they appear. In almost every case the first symptom is *contraction of the muscles of the jaw and neck*, which may be so mild that a diagnosis of rheumatism is made. It soon increases, however, into tonic spasm. *Trismus*, practically always the first symptom, is quickly followed by *opisthotonus*. The *muscles of the pharynx and larynx* may be soon involved ; a feeling of great tension in the throat and *difficulty in swallowing* are then early symptoms. The intensity of the spasm increases from

FIG. 440.—Facial expression and general appearance in tetanus. (After Brunner.)

day to day, spreads to the *muscles of the face and trunk*, and finally to those of the *extremities*, although the arms, hands, and fingers may be altogether spared. This progress of the spasm from one group of muscles to another follows a descending direction, and shows a certain regular order. The ocular muscles are rarely involved. The patient is now in a pitiable condition, especially as the *respiratory muscles* become involved. He lies in bed, breathing with difficulty, and his suffering is portrayed in the expression of his face, the rigidly contracted features, the furrowed forehead, the eyelids drawn down by the spasm of the orbicularis, and the droop at the corners of the mouth, which is dragged to the sides (Fig. 440). The jaws are tightly closed, the head drawn backward, the abdominal muscles are as tense as boards, and the muscles stand out distinctly on the legs, which are in a position of extension. Sleep alone relaxes the muscles. In a few cases there is emprosthotonus or pleurothotonus instead of opisthotonus. This chronic spasm causes *pain*, which is sometimes very severe and becomes more acute during the *attacks*. We speak of a tetanus dolorosus. From time to time—it may be at long intervals or in rapid succession—there is a *convulsive exaggeration* of the muscular

contraction, which suddenly reaches its maximum intensity, spreads over the whole body, and causes violent pain. These attacks, which as a rule only last for a few seconds, are usually of *reflex* origin—due to the patient being touched or shaken, or to some stimulation of his special senses. The involvement of the laryngeal and thoracic muscles in the spasm may cause severe *dyspnœa*. Speech and deglutition are often difficult or even impossible. The functions of the bladder and bowels may be impaired, and there may be complete retention of urine.

The mind is hardly ever affected, and the sensibility and functions of the special senses are intact. The *temperature* is normal, or towards the end of life it may rise, sometimes to a considerable height; a post-mortem temperature of over 44·0 has been reported. Profuse *perspiration* is an almost constant symptom, and the quantity of urine is accordingly diminished, the scanty urine being very much concentrated. Albuminuria may occur, and Mostri has observed herpes zoster in the course of the disease.

With regard to the condition of the urine and metabolism in tetanus, see Senator (*B. k. W.*, 1905).

The local muscular contractions, occurring in the neighbourhood of the focus of infection, which are so characteristic of experimental tetanus, have only been observed in a very few cases in man (*e.g.* by Klemm, Halban, and others).

Duration and Course.—The disease usually lasts for several weeks.

According to Rose, five stages can be distinguished in the fully developed disease: 1. that of trismus; 2. that of general rigidity, beginning in the muscles of the neck and spreading to those of the back, abdomen, and pelvis, and finally to those of the lower limbs; 3. that of general spasms, in which there is no increase of the reflex excitability; 4. that of reflex spasms, and 5. that of exhaustion. Tetanus has been classified into different forms—acute, chronic, mild, medium, etc.—according to the acuteness of its development and the course and severity of the symptoms (Larrey, Rose).

If the disease takes a favourable course, the spasms first disappear, then the continuous rigidity yields; trismus is the last symptom to disappear. Convalescence usually requires a long time. The prospects of recovery are more favourable in the chronic than in the acute cases. In exceptional cases localised contractures may persist after recovery—*e.g.* in the trapezius in a case of Adrian's. The transition into a form of chronic tetanus—an extremely rare occurrence—has been described by Brunn and Grober, and by Demontmerot (*Thèse de Paris*, 1904). In the great majority of cases (90 per cent., according to the latest statistics) the disease ends in death, which usually occurs within five or six days, but may not take place till a fortnight later. *Asphyxia, exhaustion or paralysis of the heart*, are the immediate causes of death.

If the first week has passed, the prognosis becomes more hopeful, and after the fourteenth day recovery may be regarded as probable. If the symptoms are mild from the first, and only increase gradually and slightly, recovery may be expected. The prognosis is generally grave when the incubation period has been short, and the first symptoms appear within five days after the injury. Puerperal tetanus is a particularly severe form, but recovery has occasionally taken place (Jacob, Kraus, Wittington).

As regards the prognosis of trismus and tetanus neonatorum et infantum, see Flesch (*D. m. W.*, 1905).

The *diagnosis* is quite simple in fully developed cases. The history of the case prevents confusion with *strychninę poisoning*, in which the symptoms also appear very soon after the poisoning, the muscular contractions at once become very extensive, and the reflex convulsions are very marked. Tetanus differs from rabies chiefly in the permanent rigidity.

Hysterical opisthotonus is seldom combined with trismus, and the hysterical nature of its symptoms is always obvious.

Pathological changes, which might, on account of their constancy, be regarded as of importance, have not so far been described. Neuritic processes have occasionally been found in the injured parts of the body, and these have suggested that the poison may first produce local changes in the nerves and may subsequently extend through their tracts to the central organs. Changes in the ganglion cells of the anterior horns of the spinal cord have been found by the Nissl method in experimental tetanus by Beck, Chantemesse-Marinesco, and in particular by Goldscheider and Flatau,[1] and have also been recognised in traumatic tetanus in man by A. Westphal, Stintzing, Matthes, and Sjövall. They appear soon after the injection of the tetanus poison, and disappear under the influence of antitoxin (see below). Courmont, Doyon, and Paviot do not regard these investigations as convincing, and Lund has also failed to find these changes in the cells of the anterior horns.

In any case we may assume that the disease is due to a toxic injury of the grey matter of the medulla oblongata and spinal cord, but the direct lesion of the peripheral parts also contributes to the production of the symptoms.

Treatment.—The patient must be protected from all excitement. He should be placed in a dark, quiet room; his bed should not be shaken, and his body should be handled as little as possible. Strengthening diet is necessary, and the tube may be passed through a space between the teeth or through the nose, or nutritive enemata may be given. It is advisable to give the patient abundance of fluid, and to stimulate diaphoresis.

Chloral hydrate is the best remedy for the muscular contractions. I have cured three out of four cases under my care by the continuous use of this drug (combined with morphia). It must be given in *large doses* (as much as 3·0 g. (45 grains) per dose, up to 180 grs. or to ½ an oz. per day in adults), and the patient should be kept as far as possible in a condition of narcosis. Other physicians prefer *chloroform*. Jaksch approves of *urethan*. A number of other drugs, such as amyl nitrite, Calabar bean, and curare (Hoffmann) have also been recommended. The latter, or *curaril*, have recently been adopted again by Bergell-Levy (*Therap. d. Geg.*, 1904), 2·4 c.cm. of the preparation being at first injected. The bromides, in large doses, have also a soothing influence. Baccelli recommends the subcutaneous use of carbolic acid; he and others have found this a means of cure. The amount contained in a hypodermic syringe of a two to three per cent. solution in sterilised water is injected every two to three hours, *i.e.* eight to ten times within twenty-four hours, the whole daily dose amounting to about 5 m. Of the thirty-two cases in which this method

[1] *Fortschr. d. Med.*, 1897; *D. m. W.*, 1898.

was adopted, only one is said to have died (?). Ascoli, Benvenuti, and Cioffi agree with Baccelli, whilst others (Josias) object to the method. *Lukewarm baths* have a soothing effect, and the permanent bath has also been used. Asphyxia may be treated by artificial respiration.

Amputation on account of tetanus is very seldom, if ever, justified. The wound should be thoroughly cleaned, enlarged, and disinfected in order to destroy the tetanus bacilli and other micro-organisms, whose presence, according to Kitasato, encourages the growth of the tetanus bacillus. Caustic and cautery should be energetically used. If it has been ascertained that the poison is excreted by the kidneys, diuresis should certainly be encouraged.

Modern discoveries have led to the adoption of different methods of treatment. Behring showed (1890) that the *blood-serum* of animals immunised against tetanus has not only an immunising, but also a curative effect. We cannot here discuss the nature of the immunising process. From this curative serum the active *antitoxin* has been prepared. Behring and Knorr, and Tizzoni have manufactured preparations for immunisation and for treatment of the disease. Others have recommended that subcutaneous injection of the serum should be replaced by intracerebral (Roux-Borrel, Chauffard-Quénu, Kocher, Lexer, Letoux) or intradural injection (Jacob, Blumenthal, Leyden, Schultze). Acting upon the advice of Wassermann and Takaki, an emulsion made from the central nervous system or the brain of healthy animals has been used for injection (Krokiewicz, Zupnik, Schuster, Holobut).

There is a very large number of cases in which one or other form of antitoxin treatment has been employed. These have been collected and worked up statistically by Köhler, Engelmann, Holsti, Ascoli, Heddaeus, and specially by Steuer. According to Holsti, out of a hundred and seventy-one persons treated with antitoxin, ninety-five recovered, whilst seventy-four died. The proportion is practically similar in cases not treated by antitoxin, although others put the percentage of deaths much higher. Steuer,[1] after a very thorough and critical statistical study, has come to the following conclusions: "In tetanus, the curative serum has hardly any effect upon the symptoms present, nor can it prevent the occurrence of fresh symptoms. Early administration of the serum, within the first thirty-six hours after the onset of the disease, has no better results. No difference can be recognised in the mode of action of the various antitoxins. There are practically never any bad effects from the application of the serum. The result of prophylactic inoculation in cases where tetanus is expected to develop is very favourable, when the antitoxin is employed at an early stage. Further, when tetanus has developed, intracerebral injection of the curative serum has no more favourable effect upon the process, and on the other hand it constitutes a danger to life. Dural infusion certainly seems to be less dangerous, but is not likely to have any better effect, etc." Kentzler[2] reports successful cases. His statistics apply to 564 cases treated with serum, of which 356 (63 per cent.) recovered. Jaksch and Stadelmann, Wilms, Möllers, C. Bruns, Stintzing, and Schuckmann have stated their doubt as to the value of serum treatment. Bockenheimer[3] reports unfavourable results from Bergmann's clinic. Antitoxin treatment is of no use after the disease has commenced. The unreliability of this treatment was also

[1] *C. f. Gr.*, 1900. [2] *B. k. W.*, 1906. [3] *B. k. W.*, 1907.

emphasised by most of the speakers in the Paris Surgical Society (April 1907). Leyden and Blumenthal advocate, though with great reserve, the continuance of the serum-treatment, as it at least counteracts the poison still circulating in the blood. Reynier, Delbet, Gibb, and others report the failure of intracerebral injection. Of fifty-two patients treated in this way, thirty-three died—63·46 per cent. (Lambert). Neugebauer [1] has used dural infusion with success. See also the discussion of the Surgical Congress of 1906 upon this subject.

Veterinary surgeons also speak with great reserve of the value of serum treatment.

All the published experience goes to show that as the results of serum treatment are so uncertain, the other remedies should never be neglected (Sahli, Rose, Heddaeus). Elsässer, from Kocher's clinic, recommends that serum injection should be combined with Baccelli's method. Behring thinks it exceedingly important that the tetanus serum should be applied as far as possible at the focus of infection, or in its close vicinity, and that the anti-toxin treatment should be commenced at the latest thirty hours after the appearance of the first symptoms, as the need for anti-toxin increases rapidly with every hour. Meyer-Ransom and E. Küster [2] are greatly in favour of local, intraneural injection.

The concentration of the serum, or the amount of antitoxin units which it contains, has been so often altered from time to time, that we shall not attempt to give exact details. Behring advises that each dose of serum should contain not less than a hundred anti-toxin units.

As regards *prophylaxis*, careful treatment of a wound, according to the principles of antisepsis, undoubtedly prevents tetanus in many instances. A number of cases are certainly known in which it has developed in spite of all precautions. We have already alluded to preventative treatment with antitoxin serum (Heddaeus, E. Kraus, Leyden, Ullrich, Lucas-Championniere, Eisendrath,[3] Suter,[4] and others), but this is by no means an absolute protection (Reynier and others).

Head Tetanus; Hydrophobic Tetanus (Rose) Paralytic Tetanus (Klemm); Bulbar Tetanus (Janin)

Literature in the review by H. Neumann, *C. f. Gr.*, 1902, and more recently in the paper by Friedländer-Meyer, *D. m. W.*, 1907.

Head tetanus is probably merely a variety of general tetanus. The tetanus bacillus has occasionally been found in the pus from the wound, but more often it is absent, whilst the secretion, blood, and transudate usually prove active in inoculations. It is caused by injury to the *face* or *skull*, and very specially by wounds of the orbit, the bridge of the nose, etc. It may also be due to a carious tooth, an otitis, etc. The incubation period lasts on an average eight to nine days.

A *spasm of the facial muscles* on the side of the injury is the *first symptom*. This is followed by *trismus*, usually at first unilateral, later bilateral. The muscular contraction then spreads to the muscles of the *pharynx* and *larynx*, and finally to those of the *neck* and *throat*. In a short time the facial spasm becomes changed into facial paralysis, either

[1] *W. kl. W.*, 1905.
[2] *Verhandl. d. Deutsch. Ges. f. Chir.*, 1905; and *Therap. d. Geg.*, 1907.
[3] *Journ. Amer. Med. Assoc.*, 1905.
[4] *A. f. kl. Chir.*, Bd. lxxv.

partial or total. In a few cases the paralysis is not preceded by spasm. Spasm, *e.g.* of the orbicularis, and paresis of the other facial muscles, may be present at the same time (observation of Solmsen). Charante describes the opposite condition. There may be no facial paralysis (Brunner, Neumann, Brunn [1]). In a few cases the paralysis involves the ocular muscles of the same side. If the wound is in the middle line of the face, the symptoms of spasm and paralysis may be bilateral (Brunner). Facial diplegia was present in the cases of Bourgeois and Crouzon, and Lloyd [2] describes a similar case. There is also usually an increase of the *reflex excitability* of the pharynx, for which reason an attempt to swallow food brings on a *spasm of deglutition*. This symptom has led to the adoption of the term hydrophobic tetanus. Dysphagia, which may develop into aphagia, has been mentioned (H. Neumann). Holub and Neumann have seen involvement of the hypoglossal muscle. There may also be spasm of the respiratory muscles. Salivation occasionally occurs. In a few cases slight *hypæsthesia* and even hyperæsthesia have been found in the face, but the sensibility is usually intact. Electrical excitability is either normal or slightly increased.

The muscular rigidity may extend still further in a descending direction, or it may be limited to the area just described. An observation by Friedländer and Meyer shows that in exceptional cases the rigidity of the neck may be absent. In every other respect the symptoms are the same as those of tetanus, but when the course is acute, fever and acceleration of the pulse are not unusual (Brunner).

The *prognosis* is grave, but less so than in general tetanus. The longer the interval between the injury and the onset of the disease, the more hope is there of recovery. The illness usually terminates fatally; out of fifty-nine cases, only twenty-four recovered. Pneumonia, erysipelas, meningitis, and nephritis are the most common complications.

Nerlich found vacuolisation of the ganglion cells in the motor fifth nucleus, and in the facial and hypoglossal nuclei, and he regards these as the pathological cause of the disease. Similar changes have been described by Preobrajenski, Bourgeois, Joukowsky, Schupfer, and Kron,[3] but these consisted partly in chromatolysis of the ganglion cells, which is of very doubtful pathological importance. In other cases also, investigation gave negative results. No changes have been found in the facial nerve.

Treatment is identical with that of tetanus, and tetanus antitoxin is said to have effected a cure in several cases (Trapp, Czyhlarz, Kocher, Helferich, Holub, Guinard.[4]

[1] *B. k. W.*, 1904.
[2] *Journ. Amer. Med. Assoc.*, 1905.
[3] *Korsakoff's Journ.*, 1906.
[4] *Société de Chirurgie*, Paris, 1907; *D. m. W.*, 1907.

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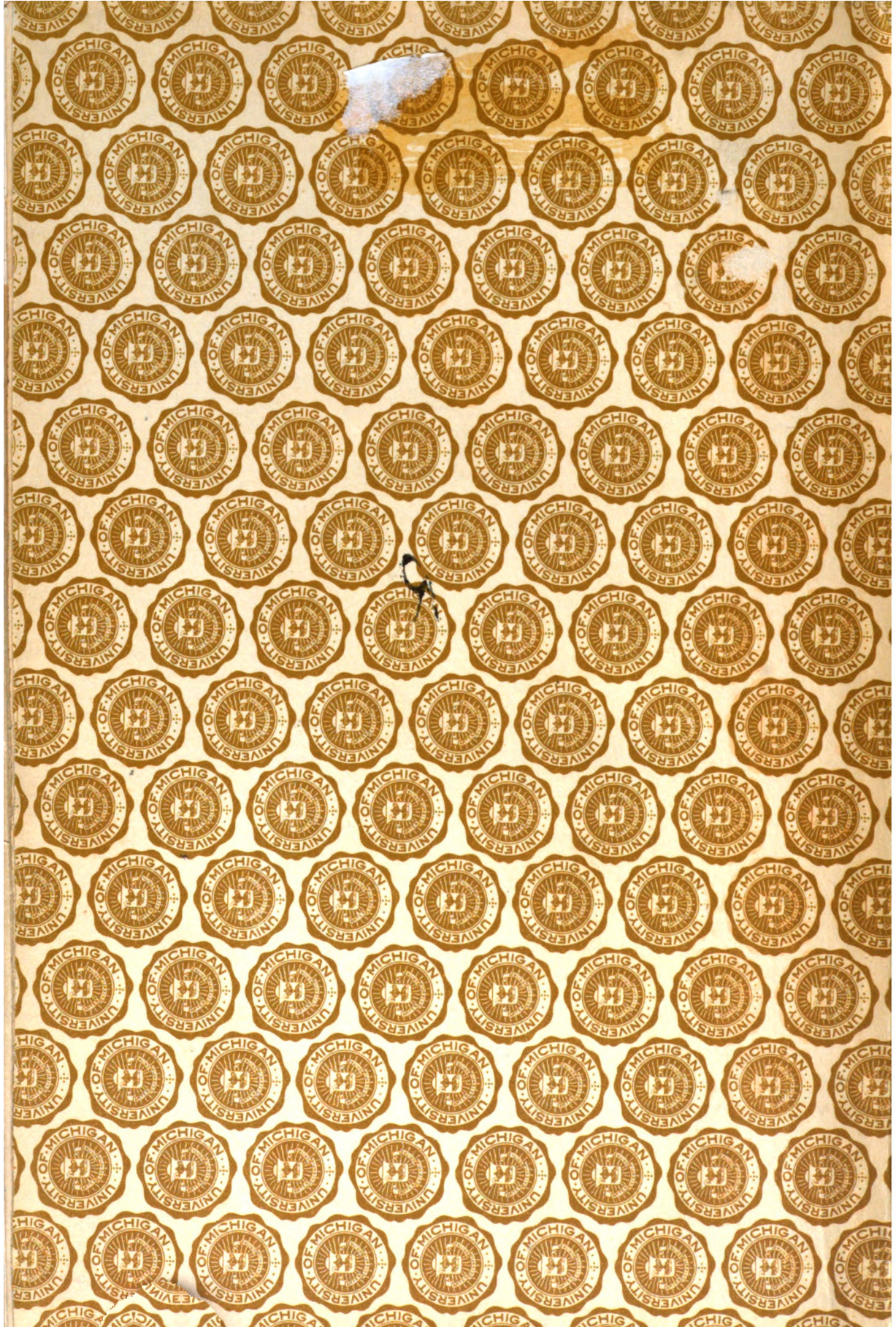
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